VOLUME 67 • MARCH 1958 • NUMBER 3

A.M.A. ARCHIVES OF OTOLARYNGOLOGY

Otitic Reinfection
George Kelemen

Studies on the Etiology of Papilloma of the Larynx
Oscar T. Pinsker and G. O. Proud

Spasm of the Cricopharyngeal Muscle as Cause of Dysphagia After Total Laryngectomy
Robert Schobinger

The Management of Malignant Neoplastic Disease Involving the Maxillary Antrum
S. Gordon Castigliano and C. Jules Rominger

Pharyngolaryngeal Disturbances Due to Cervical Spondylosis
A. Laskiewicz

Acoustic Neurilemoma
Kenneth Cambon and Frederick R. Guilford

Mixed Tumors of the Palate
Fernand Montreuil

Diagnosis and Management of Parotid Tumors
Samuel L. Perzik

Rhinology: 1900 to 1910
Maurice H. Cottle

Sinusitis and Nonspecific Endogenous Ocular Inflammation
Joseph Lubart

Alcoholic Beverage, Nasal Temperature, and the Common Cold
Noah D. Fabricant

Psychogenic Vertigo
Burness E. Moore and Miles Atkinson

Premedication in Clinical Audiometry
Raymond B. Strauss

Congenital Anomaly of Nasal Structure
Michael S. Horowitz

Nonchromaffin Paraganglioma of the Middle Ear
Thomas R. Cox, Walter Krohn, and Luis E. Remus

Chemodectoma of the Trachea
Julius W. McCall and Farid K. Karam

Endoesophageal Skin Graft in Corrosive Strictures
Blair Fearon and A. W. Farmer

News and Comment

Books

Directory of Otolaryngological Societies

AMERICAN MEDICAL ASSOCIATION PUBLICATION

5 NEW BOOKS *in this field*

of special interest to you!

ATLAS OF ESOPHAGEAL MOTILITY IN HEALTH AND DISEASE

by **Charles F. Code,** *Mayo Clinic*

A pictorial presentation of physiological mechanisms involved in the esophagus. **"The pictures tell the story."** Pictorial material flows from basic recording methods through to tests or observations of healthy individuals and then proceeds into the development of the knowledge pertaining to the derangements of motility which are produced in the esophagus by disease. The written word follows the same pattern but the two are not directly tied. The reader is not forced to turn from one to the other by cross references. This unique format enables the reader to pick up most of the information in the book by looking at the illustrations. *Pub. '58, 156 pp., 69 il.*

THE DIAGNOSIS AND TREATMENT OF INFECTIONS

by **D. Geraint James,** *Middlesex Hosp., London*

The most **up-to-the-minute techniques** available in one text concerning the rapid advances in specific chemotherapy and more precise methods of diagnosis. For handy reference informative tables are provided to complement the text. Presents the subject matter in three sections: The first provides details of **currently used chemotherapeutic agents;** the second describes **micro-organisms** responsible for human infections, and the third takes up **infections of the various systems.** *Pub. '57, 244 pp., 1 il., 24 tables, Cloth, $6.00*

ROENTGENOLOGY OF THE CHEST

Sponsored by The American College of Chest Physicians

Edited by **Coleman B. Rabin, M.D.**

The big new book they're all talking about. Eagerly awaited because of **the excellence of its 40 contributors,** including Rigler, Peabody, Sr., Swenson, Ochsner, Blades, Banyai, Schwedel, Steinberg, Medelman, de Abreu, Kane, Farinas, Westermark, Garland, Goldman, Gordon, Head, McKinlay, Touroff, Myers, Trimble, Furculow, Winn, Peabody, Jr., Sander, Kerley, Rabin, Friedman, Felson, Levine, Fleischner, Carter, Master, Dack, de Carvalho, Dotter, Gasul, Brahms, and Reich. *Pub. '58, 504 pp. (8½ x 11), 247 il., Cloth, $19.50*

UNEXPECTED REACTIONS TO MODERN THERAPEUTICS

by **Leo Schindel,** *Jerusalem*

Incorporated in this volume is material gathered from hundreds of publications from all parts of the world exploring the side-effects and untoward reactions of antibiotics. Its purpose is to make these reactions more easily prevented as more becomes known about them. The general practitioner as well as the specialist will find the book of immense interest and value. *Pub. '57, 160 pp., Cloth, $3.00*

MOULD FUNGI AND BRONCHIAL ASTHMA

by **P. J. van der Werff,** *Amsterdam Clinic for Allergic Diseases*

A mycological and clinical study based on the author's long experience in fungus allergy. This **detailed and accurate** study of the occurrence of spores of moulds in the atmosphere, indoors in houses and factories as well as the open air, presents a reliable and comprehensive survey valuable to all investigators. *Pub. '58, 250 pp., 68 il.*

CHARLES C THOMAS • PUBLISHER

301-327 East Lawrence Avenue

Springfield • Illinois

TABLE OF CONTENTS

VOLUME 67 MARCH 1958 NUMBER 3

ORIGINAL ARTICLES

PAGE

Otitic Reinfection
George Kelemen, M.D., Boston 257

Studies on the Etiology of Papilloma of the Larynx
Oscar T. Pinsker, M.D., and G. O. Proud, M.D., Kansas City, Kan. 268

Spasm of the Cricopharyngeal Muscle as Cause of Dysphagia After Total Laryngectomy
Robert Schobinger, M.D., New York 271

The Management of Malignant Neoplastic Disease Involving the Maxillary Antrum
S. Gordon Castigliano, M.D., and C. Jules Rominger, M.D., Philadelphia 276

Pharyngolaryngeal Disturbances Due to Cervical Spondylosis
A. Laskiewicz, M.D., London 292

Acoustic Neurilemoma
Kenneth Cambon, M.D., and Frederick R. Guilford, M.D., Galveston, Texas 302

Mixed Tumors of the Palate
Fernand Montreuil, M.D., Montreal 313

Diagnosis and Management of Parotid Tumors
Samuel L. Perzik, M.D., Beverly Hills, Calif. 319

Rhinology: 1900 to 1910
Maurice H. Cottle, M.D., Chicago 327

Sinusitis and Nonspecific Endogenous Ocular Inflammation
Joseph Lubart, M.D., New York 334

Alcoholic Beverage, Nasal Temperature, and the Common Cold
Noah D. Fabricant, M.D., Chicago 344

Psychogenic Vertigo
Burness E. Moore, M.D., and Miles Atkinson, M.D., New York 347

AUDIOLOGY SECTION

Premedication in Clinical Audiometry
Raymond B. Strauss, Ph.D., Cleveland 354

CASE REPORTS

Congenital Anomaly of Nasal Structure
Michael S. Horowitz, M.D., London 364

Nonchromaffin Paraganglioma of the Middle Ear
Thomas R. Cox, M.D.; Walter Krohn, M.D., and Luis E. Remus, M.D., Dallas, Texas 368

Chemodectoma of the Trachea
Julius W. McCall, M.D., and Farid K. Karam, M.D., Cleveland 372

CLINICAL NOTES, NEW INSTRUMENTS AND TECHNIQUES

Endoesophageal Skin Graft in Corrosive Strictures
Blair Fearon, M.D., F.R.C.S. (C), and A. W. Farmer, M.D., Toronto 374

REGULAR DEPARTMENTS

News and Comment 381

Books 383

Directory of Otolaryngological Societies 385

A. M. A.
ARCHIVES OF OTOLARYNGOLOGY

VOLUME 67 MARCH 1958 NUMBER 3

COPYRIGHT, 1958, BY THE AMERICAN MEDICAL ASSOCIATION

EDITORIAL BOARD

GEORGE M. COATES, Chief Editor
1721 Pine Street, Philadelphia 3

RALPH A. FENTON, Portland, Ore.
ROBERT L. GOODALE, Boston
VICTOR GOODHILL, Los Angeles
CHEVALIER JACKSON, Philadelphia
JOHN D. KERNAN, New York
CARL H. McCASKEY, Indianapolis
JOSEPH SATALOFF, Philadelphia
ERNEST M. SEYDELL, Wichita, Kan.
GEORGE E. SHAMBAUGH JR., Chicago

AUSTIN SMITH, Editor, A. M. A. Scientific Publications
GILBERT S. COOPER, Managing Editor, Specialty Journals

SUBSCRIPTION RATES

Price per annum in advance, including postage: Domestic, $14.00. Canadian, $14.50. Foreign, $15.50. Price to students, interns, and residents, $8.00 in U. S. & possessions.

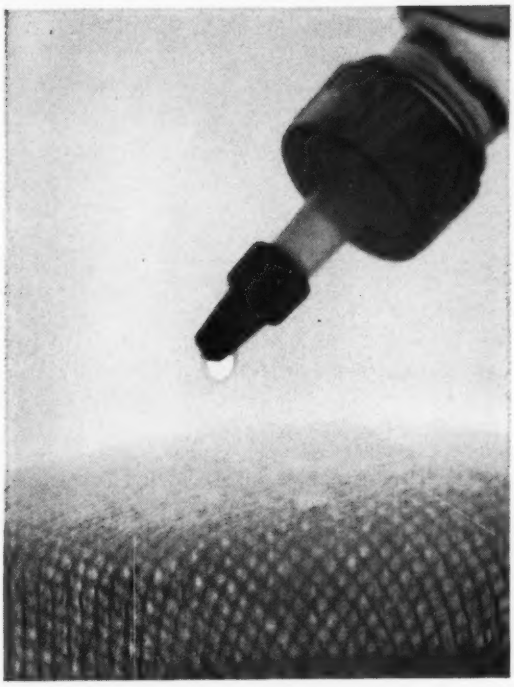
Single copies of this and previous calendar year, $1.50.

Back issues older than two years are available through Walter J. Johnson, Inc., 111 Fifth Avenue, New York 3. Future reprints of back issues will be available through Johnson Reprint Corporation, 111 Fifth Avenue, New York 3.

Checks, money orders, and drafts should be made payable to the American Medical Association, 535 North Dearborn Street, Chicago 10.

AMERICAN MEDICAL ASSOCIATION Publication

Published monthly by the AMERICAN MEDICAL ASSOCIATION. Editorial and Circulation Offices: 535 North Dearborn Street, Chicago 10, Illinois. Publication Office: Thompson Lane, Box 539, Nashville 1, Tennessee. Change of Address: Notice to the circulation office should state whether or not change is permanent and should include the old address. Six weeks' notice is required to effect a change of address. Second-class mail privileges authorized at Nashville, Tenn., Aug. 6, 1956.



For smooth, peaceful induction

VINETHENE®

(VINYL ETHER FOR ANESTHESIA, U. S. P.)

Rapid, peaceful induction has been achieved with VINETHENE for almost a quarter of a century. VINETHENE is excellent for short operative procedures, since it quickly effects analgesia, produces good muscular relaxation, and rarely causes excitement or nausea. May be administered via open, semi-closed or closed methods.

Supplied: In 10-cc., 25-cc., 50-cc. and 75-cc. bottles, each with adjustable plastic dropper cap.

Color Film for Professional Use—A 16 mm. motion picture film in color, entitled "Anesthesia with Vinethene in Short Operative Procedures," is available for viewings. Arrangements can be made by writing to Merck Sharp & Dohme, Philadelphia 1, Pa.

VINETHENE IS A TRADEMARK OF MERCK & CO., INC.

MERCK SHARP & DOHME

DIVISION OF MERCK & CO., INC. PHILADELPHIA 1, PA.

Instructions to Contributors

Articles, book reviews, and other materials for publication should be addressed to the Chief Editor. Articles are accepted for publication on condition that they are contributed solely to this journal.

An original typescript of an article, with one carbon copy, should be provided; it must be double or triple spaced on one side of a standard size page, with at least a 1-inch margin at each edge. Another carbon copy should be retained by the author.

The main title of an article may not contain more than eighty characters and spaces; a subtitle may be of any length.

The author's name should be accompanied by the highest earned academic or medical degree which he holds. If academic connections are given for one author of an article, such connections must be given for all other authors of the article who have such connections.

If it is necessary to publish a recognizable photograph of a person, the author should notify the publisher that permission to publish has been obtained from the subject himself if an adult, or from the parents or guardian if a child. An illustration that has been published in another publication should be accompanied by a statement that permission for reproduction has been obtained from the author and the original publisher.

Oversized original illustrations should be photographed and a print on glossy paper submitted. Prints of a bluish tinge should be avoided. Large photomicrograph prints will be reduced in scale unless portions to be cropped are indicated by the author. The author should submit duplicate prints of roentgenograms and photomicrographs with the essential parts that are to be emphasized circled, as a guide to the photoengraver.

Charts and drawings should be in black ink on hard, white paper. Lettering should be large enough, uniform, and sharp enough to permit necessary reduction. Glossy prints of x-rays are requested. Paper clips should not be used on prints, since their mark shows in reproduction, as does writing on the back of prints with hard lead pencil or stiff pen. Labels should be prepared and pasted to the back of each illustration showing its number, the author's name, and an abbreviated title of the article, and plainly indicating the top. Charts and illustrations must have descriptive legends, grouped on a separate sheet. Tables must have captions. ILLUSTRATIONS SHOULD BE UNMOUNTED.

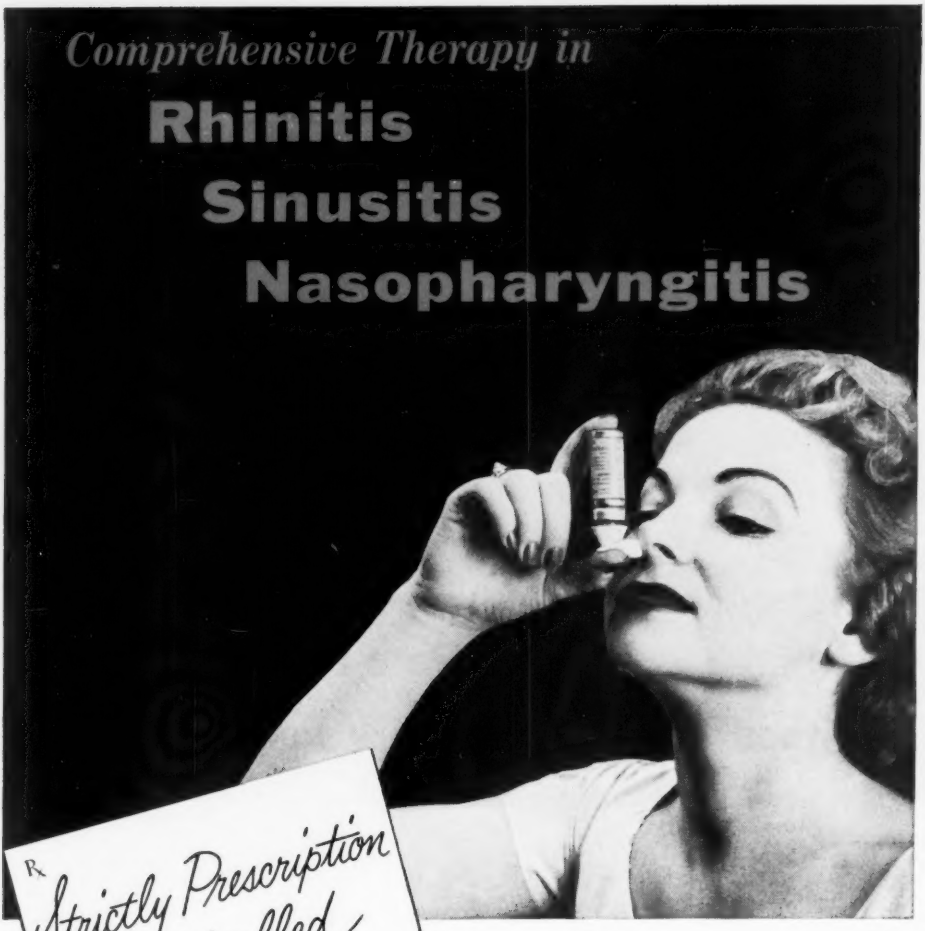
References to the literature should be limited to those used by the author in preparation of the article. They should be typed on a special page at the end of the manuscript. The citation should include, in the order given, name of author, title of article (with subtitle), name of periodical, with volume, page, month—day of month if weekly or biweekly—and year. References to books must contain, in the order given, name of author, title of book, city of publication, name of publisher, and year of publication.

AMERICAN MEDICAL ASSOCIATION

535 North Dearborn Street **Chicago 10**

Comprehensive Therapy in

Rhinitis
Sinusitis
Nasopharyngitis



℞ *Strictly Prescription Controlled*

Medihaler-Phen®

MEASURED-DOSE NASAL AEROSOL NEBULIZATION

Immediate, effective 4-pronged relief...vasoconstrictive, decongestive, anti-inflammatory, antibacterial. Accurately measured nebular cloud makes droppers and squeeze bottles obsolete.

Each cc. contains phenylephrine HCl 3.6 mg., phenylpropanolamine HCl 7.0 mg., neomycin sulfate 1.5 mg. (equivalent to 1.0 mg. neomycin base), and hydrocortisone 0.6 mg., suspended in an inert, nontoxic aerosol medium.

Riker LOS ANGELES

MEDIHALER MEANS

automatically measured-dose aerosol medications for closer management supervision over the patient.

Test More Patients Daily,

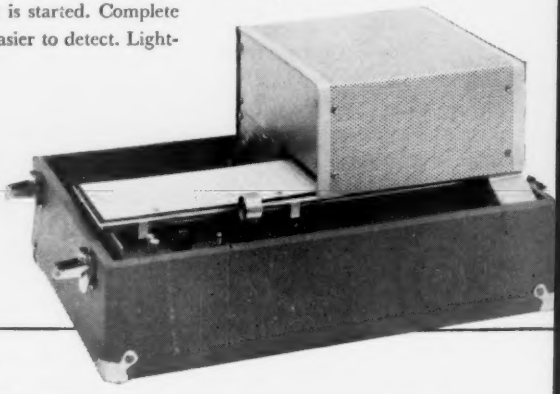
Without Extra Supervisory Help

with the new Rudmose

Automatic Audiometer

Make your own time more productive with a Rudmose Automatic Audiometer. No need to wait while hearing test is being completed. Subject conducts own pure tone test with high degree of accuracy. No supervision needed after test is started. Complete test takes about six minutes. Malingering easier to detect. Lightweight audiometer is easily handled.

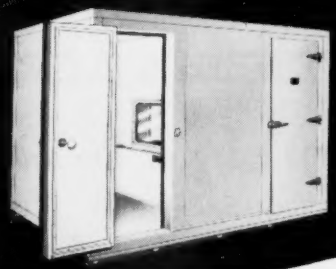
- *completely automatic*
- *saves your time, money!*
- *subject controls own test*



MAICO
ELECTRONICS INC.

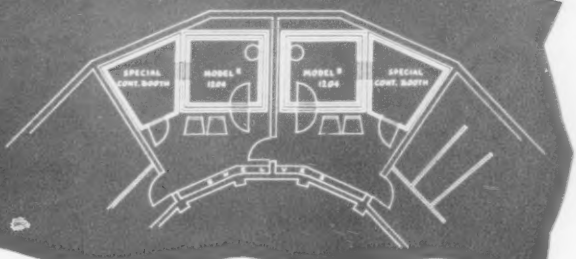
for complete details write Room 108-A
21 N. Third St., Minneapolis, Minn.

MAKERS OF
Electronic Stethoscopes • Conventional Hearing Aids • Hearing Glasses
Ear Defenders • Audiometers • Group and Classroom Hearing Aids



IAC Series 400-CT standard Audiometric Examination Room combines an Examination Area and Control Area in one unit.

standard-ized...or...special-ized



Floor plan of a projected Speech and Hearing Center to be constructed in a famous institution.

IAC Your Standard of Silence

audiometric examination rooms

provide the ultimate in required performance at lowest cost!

IAC advanced acoustical engineering makes available to the Otolaryngologist and Audiologist, Audiometric Examination Rooms with performance guaranteed by known results in hundreds of existing installations.

IAC Audiometric Examination Rooms are prefabricated in many models to meet your acoustical and structural requirements. Where special audiometric installations are desired, IAC's engineering staff is at your, and your architect's disposal, for the design, construction and installation of complete Audiology Clinics and Research Centers — at the same low cost per square foot as the standard rooms!

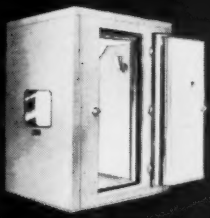
Professional literature will be furnished on request.

Medical Products Department

INDUSTRIAL ACOUSTICS COMPANY, INC.

341 Jackson Avenue • New York 54, N. Y.

Series 400

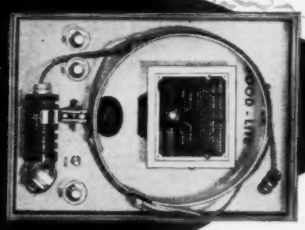


Series 1200

For Information Write Dept. O-3

new SUPER HEADLIGHT

BY GOOD-LITE



- *COOLER OPERATION*
- *9 INTENSITIES OF LIGHT*
- *INCREASED SAFETY— UNDERWRITERS APPROVED*

Medical men who have tried the new Super Good-Lite tell us there's nothing like it anywhere . . . at any price! Here's why: The entire unit has been redesigned by a group of *practicing surgeons* to provide a beam of light both brilliant and controllable . . . featuring nine intensities of light. The headlight construction and electrical circuit was designed for cooler operation and maximum safety.

The Good-Lite SUPER Headlight Includes:

- Good-Lite parallel beam headlight.
- 3 types of bulbs with 3 intensities of light each.
- Special Ground lens to intensify light.
- 2 mirrors (peep-hole and plain).
- Heavy duty transformer (20 watt) with 3 positions and off switch. Fuse in the secondary for your safety.
- Molded plastic connections.
- Gray plastic stainproof cords.
- Choice of 5 types of headbands.

Consult Your Surgical Supply Dealer or Send Coupon Today

GOOD-LITE MFG. CO.
7638 W. Madison St. • Forest Park, Ill.

Please send free catalog on Good-Lite Headlights to:

Dr. _____

Address _____

City _____ Zone _____ State _____

FULL-RANGE FOCUSING SPEED-ILLUMINATOR

A MOST USEFUL Lighting Instrument

DIAGNOSIS – TREATMENT – MINOR SURGERY –

Ask your dealer for:
Head Line #106-C..... $22.50
Spare bulbs 4 for only... 1.50

- Unrestricted maneuverability
- Projects brilliant cool-beam
- Color controlled for accurac
- Focus from large to tiny spo
- Universally positionable hea
- Finest optics. NO reflector
- Always bright. NO batteries
- Safe, Shock-Proof transform
- Operates from standard A.C
- Convenient built-in switch
- Compact. Surprisingly light
- Built to serve a lifetime
- Low cost, low voltage bulb
- NOT a flashlight

The HEAD LINE Co. • Flushing 66, N.Y.

YOU CAN DO A COMPLETE

TRACHEOTOMY IN SECONDS

The Sierra-Shelden Tracheotomy Set consists of a

Slotted Guide Needle,

a Tracheotomy Tube,

a Cutting Trocar which is positioned by the Slotted Guide Needle,

and a plastic Inner Cannula.

in sizes 4 and 6 through surgical supply dealers

with the new

Sierra®

SHELDEN TRACHEOTOME

A tracheotomy, whether elective or emergency, can be performed in less than 30 seconds with this new precision instrument. The fast, accurate technic is equally effective in a hospital or in the field... reducing trauma to a minimum.

The set is packed in a compartmented cloth bag and includes plastic sheaths for trocar and needle. Refer to illustrated instruction booklet packed with each instrument before using.

Write for Medical Journal Reprint describing technic

R. A. HAWKS DIVISION Sierra Engineering Co.

123 E. MONTECITO AVE., SIERRA MADRE, CALIFORNIA

easier
antipyretic–analgesic
relief

in respiratory infections
and following tonsillectomies

you help her recover more easily
when you prescribe

Tempra

Acetaminophen, Mead Johnson

drops/syrup

Tempra is the first physician-controlled antipyretic–analgesic in two liquid dosage forms–wild-cherry-flavored Drops and mint-flavored Syrup. Both are readily accepted, well tolerated and easy to give, without forcing or fussing.

Since Tempra is on Rx only, you have better control of medication and dosage... parents have added confidence.

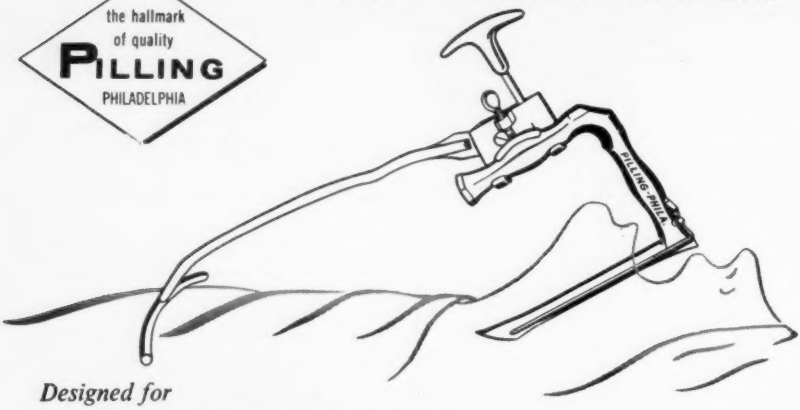
For detailed brochure on Tempra–you are cordially invited to ask your Mead Johnson Representative or write us, Evansville 21, Indiana.

Mead Johnson
Symbol of service in medicine

TE440

LEWY LARYNGOSCOPE HOLDER for use with standard JACKSON and HOLINGER INSTRUMENTS

the hallmark of quality PILLING PHILADELPHIA



Designed for

ROBERT B. LEWY, M.D., Chicago

The Lewy Laryngoscope Holder is quickly and easily attached to any standard type Jackson or Holinger laryngoscope as well as a special adaption of the Lynch laryngoscope. Thus the Lewy Laryngoscope Holder permits relaxed laryngoscopy by converting a standard laryngoscope into a self-retaining unit.

The combination sprocket and worm gear maintains complete exposure of the vocal cord at all times. Even in the most difficult cases, the non-slipping gear power affords the laryngologist two free hands. He can now operate without the strain of manually holding the instrument and accomplish careful unhurried work. Furthermore, removal of neoplasms becomes more accurate and thoroughness of examination is thus improved.

In positioning the patient, under-the-shoulder support is sufficient. However, if desired, the patient's head may be held by an operating room assistant or a mechanical head holder.

A special adaptation of the Lynch laryngoscope may be used with the Lewy Laryngoscope Holder, eliminating the need for the Killian Gallows. One special long spatula and pair of tooth plates, as illustrated, are supplied. Finish is black.

Reference—R. B. Lewy: Acta Oto-Laryngologica, Vol. 46, Fasc. 1, 1956

Br248 LEWY LARYNGOSCOPE HOLDER	$79.50
Br248-A LEWY LARYNGOSCOPE HOLDER with hinged handle	$86.00
Br249 LYNCH LARYNGOSCOPE modified for use with Lewy Holder	$125.00

JACKSON AND HOLINGER LARYNGOSCOPES—write requesting information

Prices f.o.b. Philadelphia and subject to change without notice.

Order these instruments direct from Pilling

GEORGE P. PILLING & SON COMPANY

3451 Walnut Street, Philadelphia

HEADQUARTERS FOR INSTRUMENTS FOR BRONCHOESOPHAGOLOGY

Pilling New York Office—4 W. 56th St., N. Y. 19, N. Y., Circle 5-8125

George S. Weigand, 4988 Reforma Rd., Woodland Hills, Calif.

James M. Francisco, 4412 E. Broad St., Columbus 13, Ohio

CABLE ADDRESS: Surgical-Phila.

A.M.A. ARCHIVES OF
OTOLARYNGOLOGY

Otitic Reinfection

An Experimental Study

GEORGE KELEMEN, M.D., Boston

The human hearing organ can undergo microscopical examination in its entirety only once, and this post mortem. To observe intermediate states of a pathological process one has to turn to animal experimentation. In aural diseases which lead unavoidably to the end, the condition in which the ears are found corresponds to an irreversible stage. An intermediary state of ear pathology is seen only when the specimen originates from a patient who died because of an extra-aural illness; otherwise intermediary stages have to be reconstructed largely by conjecture or by forming an arbitrary sequence of findings as obtained from different persons.

Animal experimentation can follow a straight course. When the aim is to study inflammatory processes, for example, the agent introduced into a group of animals can be one known to result in certain similar lesions. By killing them in subsequent periods one can follow the course of the disease. Yet this experimental procedure, simple as it is in principle, meets formidable obstacles.

Spontaneous infections in the hearing organ of the common laboratory animals have been recognized by numerous authors. However, a far greater number of writers have reported on experimental results without paying due attention to the pathology which was present before the investigation started. Not merely can a lesion have existed before, but it may develop independently of the experimental procedure at any time in the course of the study.

Many examples of such neglect can be quoted. In one instance a painstakingly detailed description of the inner ear was given, but only a single word, and this in the summary, was considered sufficient to say that "healthy" guinea pigs were used; no data about the condition of the middle ears can be found in the paper. In other instances some attention has been paid to the tympanic cavity, as when it was revealed that "a massive otitis made microscopy difficult." In yet another instance far-reaching conclusions about isolated parts of the inner ear were accompanied by the terse remark: "the middle ears of the animals were normal"—without quoting protocols in the absence of which there could be no possibility of report on "normalcy." Such examples can be multiplied ad libitum.

Yet sufficient warning has been given: Berberich and Nussbaum (1931), Nelson and Gowen (1931), Farris and Griffith (1949), and a number of other investigators have called attention to infections in the middle ear. Nelson and Gowen found them in 40% to 50% of their rats. Aschan

Submitted for publication Sept. 4, 1957.

From the Department of Otolaryngology, Harvard Medical School, and the Department of Otolaryngology, The Massachusetts Eye and Ear Infirmary. Supported by United States Public Health Service Research Grant B-1272 (C).

(1948) was one of those who discussed this question in detail; he found in his rat material 43% infected in the middle ear and made the justified remark that infections of the upper air passages, if unheeded, constitute an equally serious source of error. Concerning such infections of the upper airways, and stressing their frequency in rat, guinea pig, and rabbit—data have been presented in a series of publications (Kelemen, 1926, 1927, 1930; Kelemen, Ambrus and Ambrus, 1955, 1958).

Berberich and Nussbaum (1931) stated that information is extraordinarily scarce on spontaneous otitis media of the current laboratory animals in spite of the fact that this condition is so frequently encountered. Induction from the external canal by parasites after perforation of the tympanic membrane is well known; otherwise infection through the Eustachian tube is the rule. Clinically, acute otitis media may manifest itself only in case of participation of the labyrinth. They mentioned that infectious rhinitis of the animals is followed by otitis media and cited instances where spontaneous cholesteatomas developed; the latter corresponded to those produced experimentally by Berberich (1927).

Nelson and Gowen (1931) discussed spontaneous otitis media in laboratory animals from many angles and found that progressing age caused increased morbidity.

Aschan (1948) put full emphasis on spontaneous otitis media in rat and rabbit, as an obstacle to experimentation with these animals. He postulated microscopical investigation, as infections will not reveal themselves in any other way in the so-called "healthy" animals. On the other hand, he was able to show that in the controls, even with inflammatory changes in the middle ear, the experimental lesions appeared to be more pronounced. Two of his statements can be fully subscribed to on the basis of our own experience: scarcity of perforations of the tympanic membrane and consequently absence of pus in the external auditory meatus as against human otitis media, and comparative scarcity of inner ear manifestations. He emphasized the wide patency of the tube in the rat; this adequate channel of drainage may reduce the frequency of tympanic membrane perforation and reduce the danger of an expansion to the labyrinth.

Friedmann (1955) found that development and character of otitis media in the guinea pig depended largely on the specific action of the infecting organism. Pseudomonas pyocyanea and Bacillus proteus caused intensive new bone formation in the middle ear. Applying the findings of Friedmann to the great number of infections encountered, the following attempt can be made to classify them on the basis of bacteriology: the quiescent, organizing processes would be attributed to Staphylococcus aureus and a β-hemolytic Streptococcus, while Diplococcus pneumoniae terminates often in death due to purulent meningitis. In case of intensive new bone formation P. pyocyaneus or B. proteus may be present.

Friedmann was right when he declared as did Aschan that suppuration can persist in the bulla for several months without manifest clinical discharge from the ear. He was right again when he underlined the observation of previous writers that new bone formation starts very early. This is to be expected, as the lining of the middle ear cavities, the mucoperiosteum, receives the impact in toto and reacts to it with all its layers—among these with the periosteum.

Contrary to the scarcity of literary data on spontaneous middle-ear infections, there is ample reporting on labyrinthitis. A point of importance often overlooked when evaluating labyrinth reactions in the presence of a middle-ear process, is a long-recognized fact, namely, in case of a purulent or even of a catarrhal otitis media in laboratory animals, there were instances with labyrinthine symptoms of high intensity without conspicuous pathohistologic findings within the labyrinthine spaces. I shall cite two examples: Marx (1910, rabbit and mouse)

and Kallberg (1939, rabbit). Both observed in the presence of a purulent, respectively catarrhal otitis media, most intensive vestibular symptoms, while the internal ear under the microscope showed practically normal conditions. Infection occurs in the first days, almost first hours, after delivery in the animal farm. Only when studying embryos can one feel perfectly safe from this danger.

Several suggestions were presented to exclude the factor of infection. Trying to produce a strain free of infections after a long line of generation is too cumbersome, especially for clinical laboratories. X-ray examination to reveal changes in transparency in the ear and the paranasal sinuses is useful in the rabbit only in a restricted way and is largely excluded in the smaller animals like guinea pig and rat. We were unable to reproduce, at least in routine examination, the pictures so convincing in the publication of Friedmann (1955). This was especially the situation when the x-ray film was compared with the findings under the microscope. Suppurative discharge from the external ear may announce a process limited to the external acoustic meatus, a favorite site of a host of parasites. On the other hand, middle-ear suppuration will often fail, as stated above, to disclose itself by aural discharge; frequently it does not produce a tympanic perforation, or the latter is already healed and remains unrecognizable under the microscope.

The present study has tried to circumvent these numerous difficulties by a different procedure—a kind of compromise. This was to take the characteristic organized residua in the middle ear as zero line and to create, by artificial impact, lesions of an undoubtedly recent nature. An interpretation then becomes possible which opposes the result of the known recently administered experimental attack against the previously existing situation. This procedure reverses, in a sense, the usual course of investigation. While generally one tries to trace the path starting from an acute condition until products of organization appear, here one differentiates the "overlay" of the later impact on the previously developed and by now mostly quiescent picture of the previous damage. The initial attack is farther removed with the increasing age of the animal. Infection starts practically at once after birth, although it may strike as a first or a repeated attack at any postnatal period. It is a serious drawback with experiments of longer duration that infection may occur during the period of study and make the condition of the experimental subject entirely different at the beginning and at the end of the investigation.

To gain a solid basis of comparison it is proposed to outline clearly the background picture as seen in a high percentage of the animals used in any particular project as experimental material or as controls. This is done in the conviction that so far the only safe way to reach valid conclusions seems to be histological scrutiny of every single animal which underwent experimental procedures, such as physiological, pharmacological, or behavioral ones, with rejection of the individuals showing old pathology from the final tabulation of results. On the other hand, when loss of valuable material in terms of time or expense may result or when otohistological analysis is only a part of the general investigation, it may be prevented by this method.

The following description will point out analogies with well-known pathohistological pictures as taken from human pathology. This has to be done, as detailed reports relating to experimental animals are relatively rare in comparison with the wide experience in human postmortem findings.

The material forming the basis of this description is compiled from a wide variety of investigational series; the illustrations, however—for the sake of uniformity—are taken from experiments with poisoning (Kelemen, Ambrus and Ambrus, 1955 and 1957) by fluoroacetate and by arsenic. It was pointed out that using drugs as tools

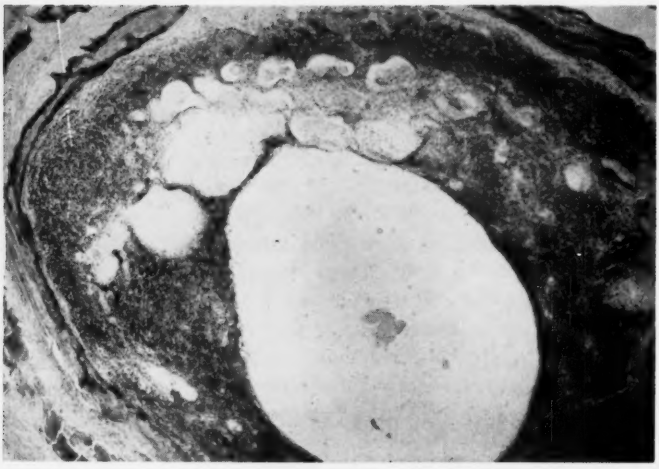


Fig. 1.—Wall of bulla (tympanic cavity). Bony capsule intact; mucoperiosteum enormously thick and containing cysts in typical "rosary" formation; free lumen much reduced. Hematoxylin-eosin: reduced about 39% from mag. × 50.

Fig. 2. — Stapes with stapedius muscle, inclusive tendon, and stapedial artery. Niche filled with solid cushion showing typical cyst formation. Functional effect would be complete immobilization. Hematoxylin-eosin; reduced about 39% from mag. × 50.

Fig. 3.—Cholesteatoma lamellae shed by the thick, granulating wall (above) and mixed with pus in the free lumen. Hematoxylin-eosin; reduced about 3% from mag. × 100.

Fig. 4.—New bone in the depth of the thickened, fibrotic mucoperiosteum, with osteoblast seams. Heidenhain-Mallory, reduced about 39% from mag. × 100.

one might elucidate, through artificially inflicted damage, questions concerning physiology and pathology of the ear. All illustrations show conditions as found in the rat. It seems appropriate to discuss the changes in the soft tissues and in the bone separately, first in the middle and then in the internal ear.

In the middle ear the most characteristic feature is the thickening of the mucous membrane to many times the normal (Figs. 1, 6, 7, 9, 10, 11). This can go so far that cushions originating from opposite walls meet in the center. A free lumen may be present merely in the form of a narrow slit (Fig. 7). The epithelium becomes more conspicuous with extensive areas converted into the high cylindrical type. Manasse (1917) stressed the point that with this transformation the mucosa loses its smooth surface and becomes uneven, with many more or less substantial protuberances but all covered with epithelium. No ulcerations are seen.

The main role in thickening of the mucous membrane falls to the subepithelial parts. The changes present two distinctly different types: granulation tissue and fibrotic tissue. The older the organizing process the greater the preponderance of the latter type. Granulation tissue shows scant intercellular substance and intensive cellular

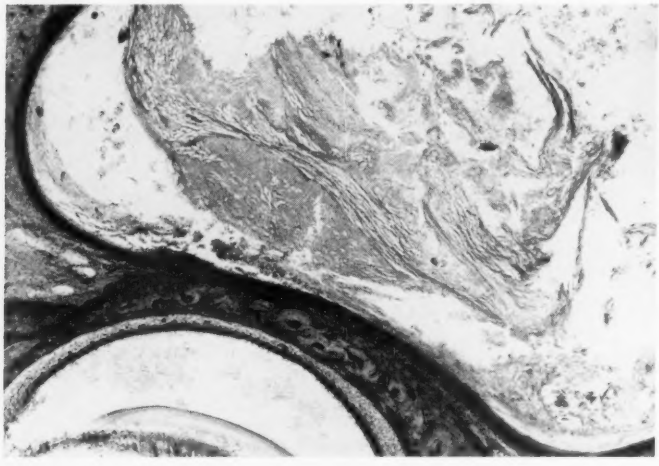


Fig. 5.—Serous labyrinthitis. Tympanic membrane adherent to promontorial wall; in external acoustic meatus ceruminous mass. Heidenhain-Mallory; reduced about 40% from mag. × 50.

Fig. 6.—Cochlea with old organized fibrous masses in all turns. Engorgement and extravasation of recent origin are added to the residua. Heidenhain-Mallory, reduced about 39% from mag. × 75.

infiltration besides ample vascularization. Old cases show a lighter-staining, fibrotic composition, poor cell content, reaching finally the state of sclerosis. Most frequently a mixture of both types will be encountered; different blocks of tissue are placed side by side or the two strata are combined in a circumscribed area of the submucosa (Figs. 1, 4, 11). Like "tree rings" these planes indicate attacks of different periods or of possibly different bacterial nature.

Nothing is more conspicuous in a similarly changed submucosa than the appearance of innumerable cysts. Their formation, the true basis of the organizing process, is not initiated, according to Manasse (1917), before at least two or three months after the acute infection. Their presence can therefore be used to "date" the process. They are never seen in acute conditions. They represent small cavities in the submucosa, in cross section round or elliptical, bounded by a thin epithelium and containing inspissated secretion. They originate from two or several processus of the submucosa, covered by epithelium, the tips of which grow together; the inner wall then forms the epithelial lining of the cyst, with frequent transformation into goblet cells. The epithelium continues to produce mu-

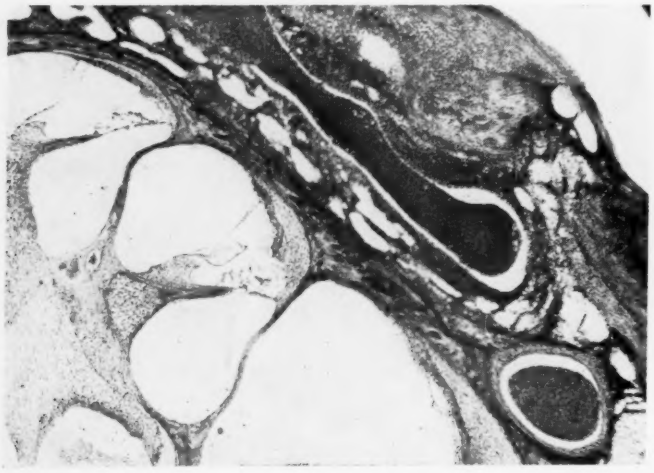


Fig. 7.—Lumen of the tympanic cavity narrowed to a slit by granulous-cystic proliferation of the mucoperiosteum. It is penetrated by a fresh abscess; no inflammatory reaction in adjacent part of cochlea; organ of Corti poorly preserved, but normal height of its mound shows that it was intact before fixation. Heidenhain-Mallory; reduced about 39% from mag. × 75.

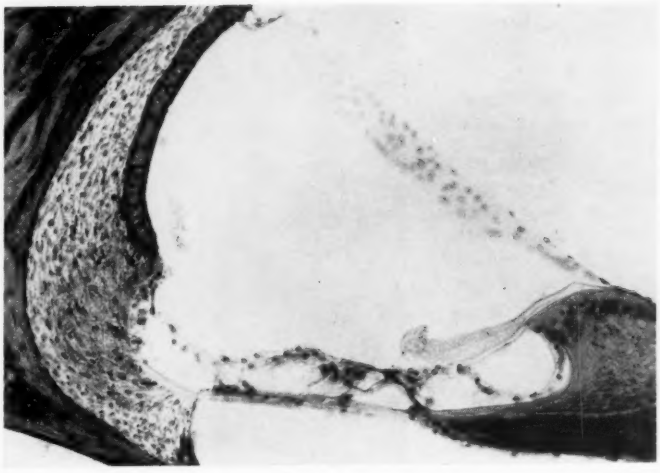


Fig. 8.—Intact organ of Corti, in parafrontal projection; Reissner's membrane in normal position. Observed alone, the normalcy of this picture could validate the result of some experiment. However, it should be confronted with Figure 9. Heidenhain-Mallory; reduced about 41% from mag. × 200.

cus, the source of further enlargement of the cyst. The processus grow to smaller or larger cushions. Ultimately they produce a very characteristic "rosary-like" picture (Figs. 1, 2, 6, 7, 8, 9, 10, 11). The cushions grow out to form polyps with predilectional sites on the promontory and in the hypotympanum.

Engorgement of the vessels is prominent in the granulation stage and poor in the fibrotic one. No extravasates are found in chronic residua.

The tympanic membrane participates in these changes, and its tympanic lining becomes similar to that seen in other parts of the cavity.

At their base the cysts are connected with the original mucous membrane by many bridges. By this process bands are produced, forming manifold connections between the walls and the ossicles and in the niche of the oval window (Figs. 2, 12). They fill the obturator foramen around the stapedial artery, which is persistent in these animals. The crura are firmly attached to the walls of the niche with resulting immobilization.

It is difficult to class cholesteatoma development among the acute or chronic fea-

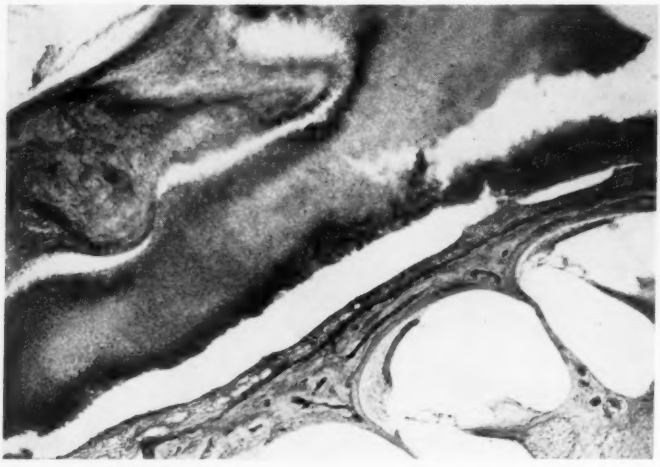


Fig. 9.—General survey of the section which contained the detail shown in Figure 8. Cystic promontorial mucosa; pus fills the tympanic lumen. To consider the findings in the inner ear alone, disregarding the general frame, would be incorrect. Heidenhain-Mallory; reduced about 41% from mag. × 100.

Fig. 10.—Pus penetrating the thick mucoperiosteum, the latter showing cystic residua. Heidenhain-Mallory; reduced about 41% from mag. × 200.

Fig. 11.—Pus from the central abscess penetrates a larger cyst of the chronically thickened mucoperiosteum by burrowing under its epithelial cover. Figures 10 and 11 show the mechanism of colliquation of old solid products in case of a reinfection. Hematoxylin-eosin; reduced about 39% from mag. × 100.

Fig. 12.—Engorgement and extravasation in the mucoperiosteum after the recent attack; the organized fibrous mass still persisting in tympanic lumen. Heidenhain-Mallory, reduced about 41% from mag. × 125.

Fig. 13.—Hyperemia in the larger basal vessels of the submucosa and in the finer vascular network of the granulation tissue announces the new attack; gradual dissolution of the mass filling the central lumen. Heidenhain-Mallory, reduced about 38% from mag. × 75.

tures, as it may appear in the course of a few weeks (Fig. 3). From the viewpoint of the present discussion this is of little significance, as cholesteatoma formation often goes on to induce labyrinthitis and quickly kills the animal (Kelemen, 1926 and 1930). Its appearance eo ipso terminates the usefulness of the animal to experimentation.

Exudate present in the free spaces of the tympanic cavity (Figs. 3, 6, 7, 10, 11) contains many kinds of cells which figure equally amid the tissue layers: small lymphocytes, leukocytes, spindle cells, stellate elements, plasma cells. Acicular spaces are left behind as phantoms of cholesterol crystals. These may be signs of degeneration without pointing to cholesteatoma formation.

Turning to changes in the bone, it should be recalled that it participates in the inflammatory process from the very beginning. In our material chronic osteitis presented itself only in its productive form (Fig. 4). The counterpart, destruction, occurred in rare instances and then only in circumscribed areas. But diffuse thickening of the osseous tympanic wall was by no means a characteristic feature. In suppurative masses osteophytes were occasionally present. The wall of the middle ear was never fistulized. This, in combination with the intact oval ligament, explains the absence of suppurative labyrinthitis in spite of severe processes in the middle ear. If massive propagation to the inner ear occurs, as reported in several instances, the animal will be eliminated from the farm population by rapid death.

The new bone is formed by osteoblasts; osteoid linings can be followed into the preformed vascular channels of the bone. Metaplastic bone is seen within the granulation tissue in the form of exostoses or hyperostoses. Hardly ever does eburnation, so well known from human pathology, appear.

Nonexperimental inflammatory changes in the internal ear occur much less frequently than in the middle ear. Generally only a collateral serous labyrinthitis appears in the wake of a spontaneous middle ear suppuration even when the latter ended in cholesteatoma. Serous labyrinthitis (Fig. 5) is hard to discover by functional tests except when a lesion has become severe. Suppurative labyrinthitis following cholesteatoma of the middle ear betrays itself by changes in posture and movement and kills the animal quickly. Although there are instances of survival after reversion of the process in the inner ear (Fig. 6), to use an animal which has passed through a similar ordeal is out of the question. Disregard

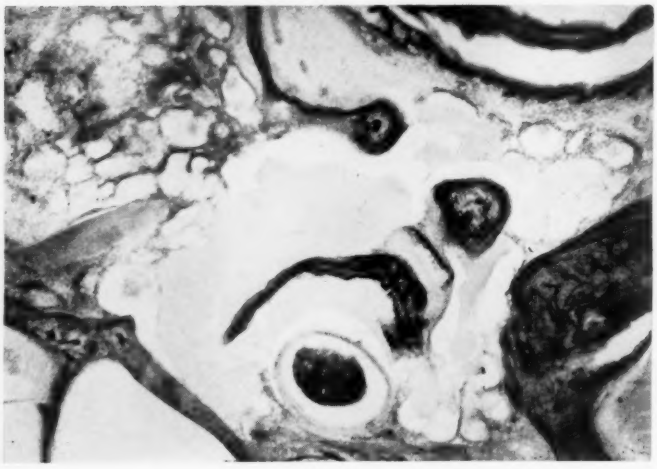


Fig. 14.—In the niche of the oval window the cystic cushion is in the process of colliquation; transudate of recent origin breaks open the walls of the cysts, destroys adhesions. Hematoxylin-eosin; reduced about 39% from mag. × 50.

of serious forms of inner ear infection renders valueless any observations concerning the function of the sensory end-organs.

All these details combine to create the picture of a chronic suppurative productive (metaplastic) otitis media complicated by a collateral serous or serofibrinous otitis interna. Against this background, then, one will have to project the products of the new experimentally inflicted damage.

Hemorrhage: Engorgement and extravasates will be found in all parts of the middle and the internal ear. Abscesses occupy the lumen of the middle ear so far as it has been left free by the organized cushions (Fig. 7). They reveal themselves as pure accumulations of lymphocytes and leukocytes without any structural organization, without connective tissue, and without vessels, showing that intra vitam they represented liquid material. A cyst-like patch of mucus or a nest of fibrin may be enclosed by the purulent mass. The pus is by no means inert. This is vividly demonstrated when it burrows under the surface epithelium with propagation of the suppuration into the infraepithelial layers (Figs. 8, 9).

The old organized residua are either able to withstand the new impingement (Figs. 10, 11), in which case the two pictures are juxtaposed or the acute abscess breaks into an old cyst and starts a fresh sequence of pathological events. With colliquation of organized products, such as bands and fibrous masses, motility of the ossicular chain may, at least to a certain degree, be restored (Fig. 12).

Even with the fresh impact of suppuration in the middle ear, the inner ear frequently remains intact (Figs. 2, 5, 7, 12). Unfortunately, nothing will betray the degree of its participation before a suppurative labyrinthitis develops.

Comment

The pathological pictures have been described in detail because they show in appearance and sequence differences contrary to conditions seen in humans. Although no experiments can be performed in men, experimental comparative pathology can hardly manage without this correlation.

It has been shown here that the important process of reinfection can be reproduced in animal experimentation. The method applied here was chemical impact. The recent attack can destroy the entire soft tissue apparatus of the middle ear, inclusive of fibrous bands and fibrous sheets. Masses occluding the oval window or forming sheets around the ossicular articulations may disappear (Fig 12).

To destroy synechiae as otitic aftermath by injecting colliquating fluid into the middle ear was tried many decades ago without appreciable success. Surgery is used to correct the conditions caused by these products of inflammation. Yet nothing can change the picture so radically as a reinfection, after which new organization may appear, possibly in a lesser degree. Clinically, a corollary is offered by the patient with otitic residua who reports that whenever his ear starts to discharge again his hearing improves.

Katsuki and Covell (1953) rightly stated that there is no organ in the body for which more observations have been erroneously described as pathological than the inner ear. It might be added—in no other organ has the surrounding pathology so often been disregarded when changes in the inner ear were declared to be the results of the experiment.

Summary

Spontaneous infection is very common in the hearing organs of the current laboratory animals. Means to ascertain their condition in vivo are meager. A large percentage can be saved for investigating purposes with exact demarcation of the pathological picture as caused by chronic organizing processes. When advantage is taken of this knowledge and necessary caution is observed, these ears still can be used for "acute" experiments. Lesions as left by a recent, artificial impact will demarcate sufficiently when projected against the background of old and hitherto quiescent residua. Discrimination between the two sets of products may serve as a starting point for further analysis.

Clinically, inverting a rigid static concept into a dynamic one, one will have to remember the perpetually changing nature of residual products instead of considering them to be definitive and immutable.

243 Charles St.

BIBLIOGRAPHY

Aschan, G. K.: Aero-Otitis Media and Aerosinusitis, Acta oto-laryng. Supp. 69, pp. 1-93, 1948.

Berberich, J.: Das Mittelohrcholesteatom: Experimentelle und pathologisch-anatomische Untersuchungen, Beitr. Anat. Physiol. Path. u. Therap. Ohres 26:1-94 (Nov.) 1927.

Berberich, J., and Nussbaum, R.: Pathologische Anatomie des Gehörorgans, in Anatomie und Pathologie der kleinen Laboratoriumstiere, edited by R. Jaffé, Berlin, Springer-Verlag, 1931, pp. 495-510.

Farris, E. J., and Griffith, J. Q., Jr., editors: The Rat in Laboratory Investigation, Ed. 2, Philadelphia, J. B. Lippincott Company, 1949.

Friedmann, I.: The Comparative Pathology of Otitis Media.—Experimental and Human, J. Laryng. & Otol. 69:27-50 (Jan.) 1955; 69: 588-601 (Sept.) 1955; 71:313-319 (May) 1957.

Kallberg, H.: Anomalies statiques et motrices chez un lapin, Rev. d'oto-neuro-opht. 17:176-182 (March) 1939.

Katsuki, Y., and Covell, W. P.: The Organ of Corti by Phase Microscopy, Laryngoscope 63:1-17 (Jan.) 1953.

Kelemen, G.: Cholesteatomatöse, beiderseitige Panotitis beim Kaninchen. Arch. Ohren-, Nasen- u. Kehlkopfh. 116:71-82 (Dec.) 1926.

Spontane Labyrinthreizerscheinungen an einigen Säugetieren, Congr. Internat. Zool. 1927, pp. 710-712.

Otitis interna acuta sero-fibrinosa mit Zwangschaltung bei einem Meerschweinchen, Arch. Ohren-, Nasen- u. Kehlkopfh. 125:275-282 (May) 1930.

—Ambrus, J. L. and Ambrus, C. M.: Fluoroacetate Poisoning and the Hearing Organ, Ann. Otol. Rhin. & Laryng. 64:1046-1056 (Dec.) 1955.

Arsenious Acid and the Hearing Organ, Acta Oto-Laryng. 1958 (in press)

Manasse, P.: Handbuch der pathologischen Anatomie des menschlichen Ohres, Wiesbaden, J. F. Bergmann, 1917.

Marx, H.: Beitrag zur vergleichenden pathologischen Anatomie der Labyrinthitis, Ztschr. Ohren. 61:1-6, 1910.

Nelson, J. B., and Gowen, J. W.: The Incidence of Middle Ear Infection and Pneumonia in Albino Rats at Different Ages, J. Infect. Dis. 46:53-63 (Jan.) 1930.

Establishment of Albino Rat Colony Free from Middle Ear Disease, J. Exper. Med. 54:629-636 (Nov.) 1931.

Werner, Ch. F.: Das Labyrinth, Leipzig, Georg Thieme, 1940.

Studies on the Etiology of Papilloma of the Larynx

Progress Report

OSCAR T. PINSKER, M.D., and G. O. PROUD, M.D., Kansas City, Kan.

Papilloma of the larynx remains one of the most vexing problems facing laryngologists today. At present, the best that can be offered a papilloma patient is judicious surgical removal and the topical application of podophyllin. In those with minimal disease this procedure may result in a cure, but more often, particularly when the disease is extensive, years of laryngeal obstruction and dysphonia are to be expected. Occasionally the papillomata are implanted in the bronchi, and the prognosis becomes even more serious. More must be known about the etiology of laryngeal papilloma before the problem of therapy can be intelligently approached.

Virologists generally agree that the various papillomata which plague mankind are caused by similar, if not by identical viruses. Von Rooyen [1] categorized them as follows: Common gray firm warts, small flat juvenile warts, pedunculated digitate types, delicate filiform warts, genital papilloma, and papilloma of the larynx. Patients having several varieties of warts at the same time are commonly noted, and certainly many patients with laryngeal papilloma are seen with cutaneous warts. Not infrequently, venereal papilloma or the history of venereal papilloma is noted in the parents of infants with papilloma of the larynx.

The virus etiology of laryngeal papilloma has been definitely established by the work of Ullman [2] in 1923, confirmed by Ishikawa [3] in 1936. They both found that papillomata appeared on the vaginal and oral mucosa of dogs several months after the injection of filtrate of human laryngeal papilloma. They also injected the skin of human volunteers with papilloma filtrate, and flat warts appeared at the injection site in two to three months. The transferability of all the other types of warts from one human being to another has frequently been demonstrated. As far back as 1918, Waelsch [4] injected the skin of his own arm with serum from veneral papilloma, and reported the appearance of flat warts three months later at the injection site.

The evidence for an antibody response to papilloma is less definitive. Maderna [5] in 1935 demonstrated complement fixation between preparations of venereal warts and the patient's serum. Findlay [6] in 1930 reported that he was able to immunize himself against wart infection after three separate injections of wart material. Brain [7] states, without further elaboration, that papillomata do not cause an antibody response, and that he had no success with wart vaccines in preventing the disease.

Squamous cutaneous papilloma in rabbits has been studied extensively by Shope,[8] Greene,[9] and others. They have isolated a virus as the etiological agent of this disease, and can reproduce the disease in another animal by inoculation of infected material. When a pregnant animal is infected, the young will frequently be born with squamous papilloma, just as newborn infants may have laryngeal papilloma. In rabbits, papilloma will undergo malignant degeneration to squamous-cell carcinoma with a high degree of frequency. Greene and others have been able to transmit this disease to animals other than rabbits. We have used some of these techniques as well as some

Submitted for publication June 27, 1957.

From the Department of Otorhinolaryngology of the University of Kansas School of Medicine.

of the standard tissue-culture methods in an effort to gain more knowledge about laryngeal papilloma.

Experimental Studies

EXPERIMENT 1.—Laryngeal papillomata were cut into fragments approximately 0.5 mm. in greatest dimension. These fragments were then embedded in egg embryo clots on the wall of roller test tubes, bathed in a nutrient fluid, and placed in an incubator. Six test tubes were set up with the tissue obtained from each of four patients. Within two to three weeks slight cell growth was noted at the edges of some of the implants, but by the fourth week all of the implants had degenerated. Fluids in all of the tubes were changed every two to three days. It was hoped that when the papilloma began to degenerate, the virus would be freed. Subsequently, the supernatent fluid was harvested and then placed in test tubes containing tissue cultures of monkey kidney cells. Viruses such as those which cause poliomyelitis, the common cold, etc., will cause destruction of monkey kidney cells, so this test is used to determine the presence of virus in unknown fluid. Each of eight harvested fluids was inoculated into three or four monkey kidney cell preparations for a total of 30 tubes. In seven days, 3 were contaminated; 8 showed no destruction; 12 showed moderate destruction of cells, and 7 showed marked destruction of the kidney cells. The supernatent fluid from each of these tubes again was harvested and inoculated into fresh monkey kidney cell preparations. From a total of 28 tubes 7 showed marked destruction and 21 showed moderate destruction of the kidney cells. This can be regarded as evidence highly suggestive of the pressure of a virus in our harvested fluids.

EXPERIMENT 2.—Papilloma extract was inoculated into two different series of tubes containing strains of squamous-cell carcinoma in tissue culture. No changes as compared with the control could be observed over a period of two weeks, at which time all of the tissue cultures degenerated.

EXPERIMENT 3.—A fragment of human laryngeal papilloma was placed in the anterior chamber of the eye in five rabbits. Two rabbits immediately developed a purulent ophthalmitis; three showed no reaction. One of the infected rabbits died. The remaining four rabbits were observed for a period of three months and killed. Though the inoculated fragment was still intact, no growths or microscopic evidence of growth of the papilloma was observed.

EXPERIMENT 4.—Two complement-fixation studies were made, using papilloma extract as the antigen and serum from a 2-year-old patient with extensive laryngeal papilloma. In the first study, undiluted antigen fixed complement with the patient's serum up to a dilution of 1 to 64. However, when the antigen was diluted, complement was not fixed. This test was repeated two weeks later with fresh tissue and fresh serum. In this second test, antigen up to a dilution of 1 to 8 fixed complement with the patient's serum in dilutions up to 1 to 64.

EXPERIMENT 5.—Thirty young white mice were obtained and divided into three equal groups. Laryngeal papillomata obtained several hours previously were cut into minute fragments, and also made into an extract. In the first group of 10 mice, a fragment of papilloma was placed in the cerebral cortex. In the second, one drop of extract was inoculated into the cortex, and in the third group, fragments of mouse embryo epithelium were immersed in papilloma extract and then placed in the cerebral cortex. Two mice died as a result of the procedure. The remaining 28 were healthy at the end of three months, and when they were killed no evidence of papilloma could be found either grossly or microscopically. This experiment was repeated with another group of 30 mice with negative results.

Comment

The lysis of monkey kidney cells obtained in the first experiment is presumptive evidence for the presence of a virus. The complement-fixation tests indicate that there is an antibody response to the papilloma. We have not succeeded in growing the papilloma in tissue culture, or in reproducing the disease in experimental animals.

One can recognize several definite characteristics for the papilloma virus, and presume several others. This virus is infectious, but to a low degree. It stimulates an antibody response, but this is probably minimal and over an extended period of time. It would be logical to assume that the virus is intracellular, and that the major reaction on the part of the infected individual is local. This would certainly explain the extended course of the disease over many years, and finally the spontaneous remission which is most frequently the outcome.

University of Kansas Medical Center.

REFERENCES

1. Van Rooyen, C. E., and Rhodes, A. J.: Virus Diseases of Man, New York, Thos. Nelson & Sons, 1948.

2. Ullman, E. V.: Acta oto-laryng. 5:317-334, 1923.

3. Ishikawa, K.: Fukuoka acta. med. 29:87, 1936.

4. Waelsch, L.: Arch. Dermat. u. Syph. 124:625, 1918.

5. Maderna, C.: Riforma Med. 51:93, 1935.

6. Findlay, G. M., in A System of Bacteriology, edited by C. H. Andrews et al. London, His Majesty's Stationery Office, 1930, p. 252.

7. Brain, R. T.: Brit. Med. J. 2:1064, 1937.

8. Shope, R. E., and Hurst, E. W.: J. Exper. Med. 58:607-624, 1933.

9. Albrink, W. S., and Greene, H. S. N.: Cancer Res. 13:64-68, 1953.

Spasm of the Cricopharyngeal Muscle as Cause of Dysphagia After Total Laryngectomy

ROBERT SCHOBINGER, M.D., New York

In this communication an often insufficiently recognized cause of dysphagia will be discussed with special emphasis on its occurrence after laryngeal surgery. The disorder under consideration is characterized by a narrowing of the cervical esophagus and caused by a spasm of the cricopharyngeal muscle. The literature contains several contributions dealing with the various anatomical, roentgenographic, and clinical points of interest, which in only rare instances, however, consider the incidence of cricopharyngeal spasm after total laryngectomy.[1-23]

Material

The records and roentgenograms of all patients who in a period ranging from Jan. 1, 1955, to Oct. 31, 1956, had undergone surgery for malignant neoplasms of the intrinsic and extrinsic larynx were collected from the files of Roswell Park Memorial Institute, yielding a total of 49 patients. The various operations included 2 laryngofissures, 20 total laryngectomies, 22 combined total laryngectomy-radical neck resections, and 5 combined total laryngectomy-partial pharyngectomy-radical neck resections. Only patients with a total laryngectomy or combined total laryngectomy-radical neck resection will be considered in the present analysis. The reason for this limitation is that the anatomical alterations after total laryngectomy-partial pharyngectomy-radical neck resection must logically be considered too important as to allow any valid postoperative evaluation of the cervical esophagus in respect to spasm of the cricopharyngeal muscle. Furthermore, no postoperative esophagograms were obtained in the two cases of laryngofissure.

Accepted for publication May 6, 1957.

Incidence of Cricopharyngeal Spasm

In the group comprising the 20 patients with a total laryngectomy, normal esophagograms were registered in 8 instances. In four patients there were definite signs of spasm of the cricopharyngeal muscle. No barium swallow had been performed in five patients, and in five others this examination was carried out in the presence of a naso-

Fig. 1.—Normal cervical esophagogram after total laryngectomy.

Fig. 2.—Lateral view of esophagogram in patient exhibiting mild dysphagia after total laryngectomy combined with a radical neck resection. The posterior bulge (arrows) due to spasm of the cricopharyngeus is well outlined.

Fig. 3.—Same patient as in Figure 2. Frontal view. Arrows point to narrowing caused by cricopharyngeal spasm.

Fig. 4.—Frontal view of cervical esophagus in patient with moderately severe postoperative dysphagia. Note smooth outline of bulge (arrows).

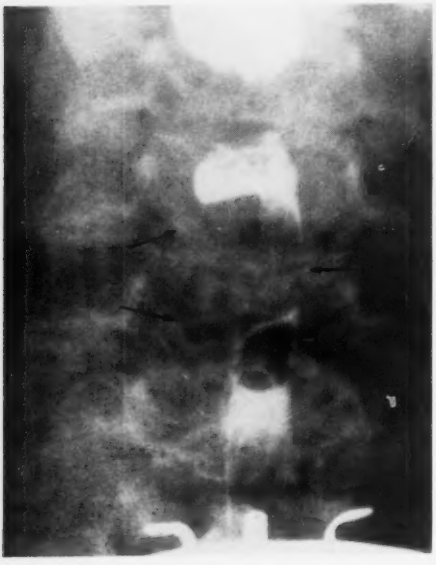


Fig. 5.—Pronounced spasm of cricopharyngeus (black arrow) combined with locally recurrent carcinoma (white arrows). Marked degree of dysphagia.

gastric feeding tube which was left in place because of clinical evidence or suspicion of a postoperative pharyngeal fistula.

Among the 22 patients who had been subjected to a combined total laryngectomy-radical neck resection there were normal barium swallows in 8 instances. In six cases, however, there existed a definite spastic constriction of the cricopharyngeus. No postoperative esophagograms were available in three patients, and in five cases a barium swallow was obtained in the presence of a nasogastric tube.

All 10 patients exhibiting roentgenographic dysfunction of the cricopharyngeal muscle complained, at the time of the x-ray examination, of variably severe dysphagia. Some patients claimed merely some mild discomfort on swallowing solid food, while some others were asymptomatic only on clear liquids. It is noteworthy that the roentgenographic intensity of the cricopharyngeal spasm did parallel the clinical severity of dysphagia in most instances (Figs. 1-5).

Comment

Crichlow submitted convincing evidence that the cricopharyngeal muscle escapes roentgenographic demonstration in normal persons.[6] An indentation of the posterior wall of the esophagus at the level of C5-C6 must, therefore, speak in favor of a spasm of this muscle. Indentations caused by osteophytes are directly located in the area of the degenerative process and can lead to diagnostic controversy only if situated at the level of C5-C6.

Preoperative roentgenograms of the esophagus were obtained in all 49 patients. The collective review confirmed normal findings in 47 instances. The two remaining esophageal examinations were on close scrutiny found to yield pathologic findings. On one occasion there was definite spasm of the cricopharyngeus associated with a typical "corkscrew" configuration of the thoracic esophagus. These findings persisted after a combined total laryngectomy-radical neck resection, and the patient continued to complain of a mild degree of dysphagia. Preoperative plain films revealed in an additional patient a small amount of air in the distal portion of the cervical esophagus. Yet the available esophagograms did not demonstrate any abnormalities of the barium column; neither did the patient complain of dysphagia. However, it was this patient who after surgery complained of progressive dysphagia and who required repeated esophageal dilatations in order to overcome his severe swallowing difficulties. The esophagoscopic examination revealed merely a pronounced narrowing of the mouth of the esophagus, which temporarily disappeared after administration of a short-acting curare-like agent. Thus, only 2 out of 10 patients who developed dysphagia and radiological evidence of cricopharyngeal spasm did exhibit preoperative roentgenographic signs of esophageal dysfunction.

None of the esophagograms obtained in the 10 patients with an indwelling nasogastric feeding tube did show clear evidence of spasm of the cricopharyngeus. This fact is brought forth only to emphasize that the presence of a foreign body in the form of a smooth plastic tube of medium caliber does not appear to foster spastic contractions of the cricopharyngeal muscle. These observations, however, do not speak against the production of a spasm of the cricopharyngeus by other foreign bodies, the rough surface of which may easily traumatize the fragile esophageal wall.[6]

In the same line of thinking, it is also difficult to associate the presence of a tracheostomy tube with the occurrence of cricopharyngeal dysfunction since the majority of the patients who were found to be free of this disorder did wear such a device.

The operative technique was in both groups of surgical procedures the same where the repair of the anterior pharyngeal wall is concerned. A T-shaped, three-layer closure had been carried out, using interrupted stitches of fine atraumatic chromic absorbable surgical suture throughout.

Progressive dysphagia accompanied by cricopharyngeal spasm of increasing intensity was observed in one case of carcinoma recurring locally after total laryngectomy (Fig. 5). Esophagograms carried out while the patient was inhaling amyl nitrite revealed no change in size of the anterior bulge (carcinoma), but a definite decrease of the posteriorly located obstruction due to spasm of the cricopharyngeus.

Hence, it is difficult to name one single cause for the postoperative occurrence of spasm of the cricopharyngeal muscle. Worthy of consideration are several factors, such as a decrease in the local blood supply, interference with the esophageal innervation, local irritation, recurrent carcinoma, metabolic disturbances or neuromuscular disorders. The last two conditions may be latent in certain individuals and become manifest in the form of cricopharyngeal dysfunction only after episodes of stress.

Conclusions

1. Spasm of the cricopharyngeal muscle must be considered as possible cause for dysphagia arising after either laryngectomy alone or combined with a radical neck resection. The incidence of this functional disorder is slightly higher in the latter surgical group.

2. The endoscopist must be aware of the possibility of cricopharyngeal spasm. Even if esophagoscopy fails to reveal any organic lesion, and thus may be reported as "negative," there nevertheless may exist a functional disorder which can be the cause of discomfort, sometimes considerable, to the patient.

3. Esophagograms may demonstrate cricopharyngeal dysfunction, but this disorder may be omitted in their diagnostic interpretation if, in the investigation of the cause of dysphagia, only organic lesions are searched for.

4. The clinical severity of dysphagia appears to parallel the roentgenographic intensity of cricopharyngeal spasm.

5. Postoperative anterior pharyngeal fistulae, plastic nasogastric tubes of medium caliber, and tracheostomy tubes did not conclusively seem to foster spasm of the cricopharyngeus.

6. Cricopharyngeal spasm may accompany locally recurrent carcinoma, and perhaps represent an early sign thereof.

7. The preoperative absence of functional esophageal disorders does not preclude the postoperative appearance of cricopharyngeal dysfunction.

130 W. Kingsbridge Road, Bronx 68, N. Y.

REFERENCES

1. Abel, W.: The Arrangement of the Longitudinal and Circular Musculature at the Upper End of the Esophagus, J. Anat. & Physiol. 47:381-390, 1913.

2. Ardan, G. M., and Kemp, F. H.: The Mechanism of Swallowing, Proc. Roy. Soc. Med. 44:1038-1040, 1950.

3. Barclay, A. E.: The Normal Mechanism of Swallowing, Brit. J. Radiol. 3:534-546, 1930.

4. Biocca, P.: Ipertrofia muscolare idiopatica dell'esofago, Arch. chir. torace. 13:275-304, 1956.

5. Crichlow, T. V. L., and Shaw, H. J.: Abnormal Ring Contractions of the Thoracic Oesophagus with an Account of Familial Manifestations, Brit. J. Surg. 42:46-53, 1954.

6. Crichlow, T. V. L.: The Cricopharyngeus in Radiography and Cineradiography, Brit. J. Radiol. 29:546-556, 1956.

7. Di Simone, G.: Studio radiochimografico dell'esofago in sogetti laringectomizzati. Ann. Radiol. Diagn. 28:372-382, 1956.

8. Elze, C., and Beck, K.: Die venösen Wundernetze des Hypopharynx, Ztschr. Ohrenh. 76-77: 185-194, 1917-1918.

9. Gray, E. D.: The Radiological Demonstration of Potential Pharyngeal Diverticulum, Brit. J. Radiol. 5:640-642, 1932.

10. Hoover, W. B.: The Pharynx and Esophagus, Surg. Clin. North America 35:629-646, 1955.

11. Hoover, W. B.: Observations on the Hypopharynx and Cricopharyngeus Area, Ann. Otol. Rhin. & Laryng. 64:874-898, 1955.

12. Hwan, K.; Grossman, M. I., and Ivy, A. C.: Nervous Control of the Cervical Portion of the Esophagus, Am. J. Physiol. 154:343-357, 1948.

13. Ingelfinger, F. J.: Disorders of Esophageal Motor Functions, Advances Int. Med. 8:11-40, 1956.

14. Jackson, C., and Jackson, C. L.: Diseases of the Nose, Throat and Ear, Philadelphia, W. B. Saunders Company, 1945.

15. Johnstone, A. S.: Non-Malignant Conditions of the Esophagus, Brit. J. Radiol. 19:101-106, 1946.

16. Killian, G.: Über den Mund der Speiseröhre, Ztschr. Ohrenh. 55:1-14, 1896.

17. Lanzara, A.: Fisiopatologia della peristalsi esofagea, Riforma Med. 70:613-619, 1956.

18. Masson, N.: Radiocinématographie de l'oesophage normal et pathologique, Ann. otolaryng. 72:928-930, 1955.

19. Mohun, M.: Globus, Tr. Am. Laryng. Rhin. Otol. Soc., 1955, pp. 142-148.

20. Negus, V.: Comparative Anatomy of the Larynx, William Heineman, Ltd., London, 1949.

21. Rogers, L.: The Treatment of Spasmodic Dysphagia by Sympathetic Denervation, Brit. J. Surg. 22:829-832, 1934.

22. Valko, P.: Segmental Spasm of the Esophagus with Laryngospasm, Česk. Otolaryng. 5:171-175, 1956.

23. Winther, K.: Spasme de l'oesophage dans le syndrome de Wallenberg, Acta psychiat. et neurol. scandinav. 30:385-394, 1955.

The Management of Malignant Neoplastic Disease Involving the Maxillary Antrum

Report of Three Cases of Total Ablation of the Ethmoid Labyrinth

S. GORDON CASTIGLIANO, M.D., and C. JULES ROMINGER, M.D., Philadelphia

The methods of management of neoplastic disease involving the maxillary antrum have fluctuated greatly during the past century. The surgical pendulum has swung from extreme radical operations with the scalpel and chisel, on the one hand, to conservative "cold surgery," electrosurgery, and irradiation delivered either pre- or postoperatively or both, and back again to pure "cold surgery."[1,7-12] The reasons for these wide changes in the same locale are diverse and important. Their influence on our methods has been as profound as elsewhere.

Our precise controlled technique utilizing intensive preoperative irradiation followed by en masse removal of the condemned irradiated maxilla and collateral tissues has evolved during the past 20 years out of the shadows of past years' experience illuminated by the brilliance of all the many advances in pre- and postoperative care, anesthesia, etc.

Historical Review

At the Oncologic Hospital the management of malignancy involving the upper jaw prior to 1929 was a more or less hit-and-miss procedure. Beginning in 1904 and extending through 1928, for the most part the management of upper jaw malignancy was an attempt at palliation rather than an attempt at cure. Most of the patients were in advanced stages of the disease. Radium applied in one manner or another played an important part in the unsuccessful treatment. Radical surgical removal of the maxilla was never attempted. The results were uniformly bad.

In 1929, a new regime took control of the Oncologic Hospital headed by the world-renowned maxillofacial and plastic surgeon, G. M. Dorrance, M.D. With his more aggressive approach, all cases of maxillary malignancy which seemed to fall within the range of technical operability were operated upon. Because of the poor results obtained in his previous experience at the Philadelphia General Hospital and the continuing poor experience at the Oncologic Hospital, by 1937 Dr. Dorrance abandoned so-called radical resection of the upper jaw as a routine procedure for the treatment of malignancy in that area. When maxillary resection was employed, it was relatively conservatively performed on cases involving the gingival structures, primarily with secondary invasion of the maxilla. Cases of primary antral malignancy or maxillary lesions with involvement of the antrum or with extension to the nasal cavity or ethmoid or orbit were all managed by means of fractionated radiation therapy. The dramatic feature of this form of treatment is the fact that it was sponsored by a surgeon who had admitted defeat in his efforts to control a disease. Dr. Dorrance was not one who readily accepted defeat.

Received for publication Aug. 29, 1957.

Read before the Section on Laryngology, Otology and Rhinology at the 106th Annual Meeting of the American Medical Association, New York, June 6, 1957.

Oncologist and Chief, Head and Neck Service, American Oncologic Hospital (Dr. Castigliano); Associate in Oncology, American Oncologic Hospital, Associate Radiologist, Misericordia Hospital (Dr. Rominger).

The fact that he conceded was strong testimony to the fact that little good was being accomplished by surgery *as performed at that time* for maxillary malignancy.

Some of the facts that we must remind ourselves of relating to those times are these. Anesthesia was almost never delivered by anyone with more than practical knowledge. The general technique of anesthesia for maxillary resection in those days was to achieve a profound stage of anesthesia, operate until the patient became too difficult to manage, then discontinue the operation until the anesthesia was again stabilized. Speed was important; technique was secondary. Fundamental oncologic concepts were often violated. Removal of tumor by morcellation was common. The employment of blood during operations was almost unheard of. The employment of antibiotics was unknown. The many secrets of postoperative care, which are so commonly accepted as routine today, were not known. So, in general, the patient in those days was operated on with a premium on speed, and finesse was something yet to be born. The incidence of postoperative complications was high. The mortality rate likewise was high. It is understandable, therefore, that one like Dr. Dorrance who had it within his power to employ any method available for the management of his patients would turn from surgery to irradiation.

Radiation therapy at the Oncologic Hospital was well advanced, considering the times. Treatment patterns were worked out employing 200 kv. radiation, a 4 gm. radium bomb, and radium or radon in an effort to control these lesions. On the basis of dosages which we today consider necessary to produce tumor-sterilizing effects in tissue, the doses delivered in that day, although lower, were within reach of the present lower carcinocidal dosages. The dosage used with the 4 gm. radium bomb even more closely approximated the lower levels of the dosages employed today. Nonetheless, good results were few and far between and complications of therapy were many and frequent, mainly due to the employment of over-sized fields and inadequate protection. Osteonecrosis produced many uphappy results.

Because of the failure to improve the results achieved by surgery through the use of fractionated radiation to relatively high dose levels, new techniques were developed, such as opening the antrum, employing radium packs, and finally resorting to repeated courses of x- or γ-radiation therapy, each course reaching relatively low levels. This was the state of affairs when one of us (S. G. C.) reached the scene in the late 30's.

After a complete review and study of the situation, by 1945 several conclusions were reached.[2] In the first place, radical surgery should be given a new trial, but the method of delivery of anesthesia would have to be improved. More careful selection of patients for surgery would be required. Efforts at attempting to devitalize the tumor by irradiation before operation should be considered. Improvements in the techniques of the performance of the operation should be evolved along with the free use of blood. Careful preoperative work-up and preparation should be carried out. A meticulous program of postoperative management should be worked out.

The above-mentioned considerations are now to be taken up in turn.

Anesthesia

Although endotracheal anesthesia was introduced in this country from England in 1929, it did not enjoy popular usage until more than a decade later. Even at this late date, except in larger clinics, endotracheal anesthesia was seldom employed because of the scarcity of physician anesthetists. Few surgeons were skilled in the technique of endotracheal administration of anesthesia.

By 1940, intravenous thiopental sodium (Pentothal sodium) anesthesia was gaining wide acceptance as a method of anesthesia

for relatively simple types of cases. In the large centers, such as the Mayo Clinic, however, intravenous anesthesia alone, or with endotracheal anesthesia, was employed in the vast majority of its head and neck operations. Movement in this direction in other clinics, and particularly in the Philadelphia area, was much slower. Several deaths in the Philadelphia area during the early 1940's, during thiopental sodium anesthesia, made it apparent that an adequate breathway must be maintained during the performance of any operative procedure. This was especially true in work involving the head and neck where laryngeal spasm could be stimulated. Pressured by events, one of us (S. G. C.) resorted to the performance of tracheotomy and the administration of oxygen through the tracheotomy opening. We have employed thiopental sodium in conjunction with tracheotomy in all of our cases requiring general anesthesia for radical surgery involving the maxilla and combined procedures involving the oral cavity, jaw, and neck. Since employing this technique, we have not had a single mortality attributed to the anesthesia or its complications. Employing this technique, we no longer fear the slippage of the endotracheal tube sometime during the course of the operation. We no longer have the anesthetist's apparatus directly in the operative area or in the head field. No longer are emergency tracheotomies necessary. In addition, the anesthetist has access to the clearing of the mucus that may develop in the major bronchi during the operation, and during the immediate postoperative course the trachea and bronchi can be kept clear. The tracheotomy tube is permitted to remain in situ for a variable period of time in our cases, depending on factors inherent in each case. In the average case the tube is allowed to remain about six days. We could not be tempted to change the technique of anesthesia as of this moment. We do not, however, choose to argue with anyone who prefers another method or who is opposed to ours.

Selection of Patients for Radical Resection of the Upper Jaw

The tendency to gauge by the size of the lesion the vigor of therapeutic attack to be brought to bear should be relegated to the past. Too often today do we still find that small lesions receive mere token radiation therapy and/or relatively small operations, while large and hopeless lesions are either overdosed by irradiation or are insulted by ultraradical, useless surgical intervention. We must avoid underbidding good hands, but by the same token we must definitely avoid overbidding poor hands. This philosophy applies with full force regardless of the situation of malignant disease.

Technical operability and categorical inoperability are terms that all cancer surgeons recognize even though there is no full agreement as to whether the base lines thus far established are to be accepted as unchangeable.[3]

Resection of the maxilla is performed for various types of malignant disease, for example, carcinoma of the upper gum, squamous-cell carcinoma and adenocarcinoma of the palate, primary malignant disease involving bone, such as fibrosarcoma, ameloblastoma, and primary malignant disease of the antrum, etc. Sharp lines of operability have not been worked out in the case of any of these lesions. For the sake of this discussion we are going to limit our remarks to primary malignancy of the antrum per se.

Advanced disease of the antrum can be considered to be present if the neoplasm has extended beyond its walls. In general, it may be said that (1) 15% of cases show extension to the orbital cavity; (2) 40% show extension to the nasal cavity; (3) 15% show involvement of the oral cavity; (4) extension to the ethmoids is difficult to prove or disprove except after surgery, but involvement is almost assured when there is bulging of the nasal wall in the region of the inner canthus of the eye; (5) involvement of the anterior cheek muscles, as evidenced by external swelling and subcutaneous induration, is present in about

15% of cases; (6) posterior extension, as can be shown by either x-ray evidence or clinical evidence, is present in about 15% of cases; (7) metastasis is present in about 20% of cases; (8) evidence of bone involvement is undoubtedly present in from 60%-70% of cases.

In our experience no sharp lines of differentiation between the inoperable and the operable case exists. Decisions as to whether a case is to be operated upon or not, with intent to cure, oftentime rests more with an over-all judgment of a particular situation, than a simple consideration of concrete data covering extent of the disease. A case operable in one surgeon's hands may be inoperable in another's. Evidence of ethmoid involvement per se does not preclude consideration of a surgical operation, but marked evidence of bone destruction with possible intracranial extension does indicate inoperability. Evidence of involvement of the orbital cavity per se does not indicate inoperability; however, marked involvement with destruction of the posterior bony plate would indicate inoperability. Extension to the contralateral side per se would not indicate inoperability, but, if the extension is marked, inoperability would be considered to be definite, and, if ethmoid involvement could be shown to be bilateral, inoperability would be definitely considered to be present. In our experience, any degree of posterior extension into the pterygomaxillary space is an indication of inoperability. Involvement of the oral cavity itself does not, in our judgment, constitute an indication for inoperability even when this extension is advanced. Cervical node involvement per se does not indicate inoperability if the extent of the primary is not advanced. On the other hand, if the prospects for cure of the primary seem dim, then the patient is generally better handled as an irradiation problem.

Not infrequently patients are seen who are considered to be borderline cases of operability. Final decision as to whether the patient should be operated upon or not depends, in our experience, on the response to a course of intensive, carefully conceived and executed precision radiation therapy. The problem of operability in the management of antral malignancy is far from being completely worked out. It is likely that as further experience accumulates we may be able to separate more precisely those patients who stand little or no chance of cure with surgical intervention.

Efforts to Devitalize Tumor Prior to Surgery by Irradiation

One of us (S. G. C.), since 1941, has been in charge of the Department of Radiotherapy at The American Oncologic Hospital and was also associated with Dr. G. M. Dorrance on the Head and Neck Tumor Service. Since 1947, he has been Chief of the Head and Neck Service. Therefore, under his control were the various methods of therapy (radiologic and surgical) required for the treatment of antral malignancy. His final decision governed the management of each case. No outside pressure influenced him as to whether radiation therapy should be employed at all or whether it should be employed as a pre- or a postoperative procedure, etc. etc. Patients, as they came to the clinic or into private practice, were treated in a completely impartial and unbiased way from a therapeutic standpoint. Armed with the previous experience, we decided that intensive precision radiation therapy should be employed as a preoperative effort to sharply defined areas. The treatment should be delivered in such a way as to make possible the surgical removal of the irradiated tissue in all but the posterior quadrant. Up to 1948, the treatment was delivered by conventional methods of treatment; after 1948, by means of supervoltage radiation generated at 2,000,000 volts.

Radiation, to be effective, must be delivered to high dosages considered to be cancer-lethal. If radiation is delivered to such high dosages and the irradiated tissues are left in situ, the undesirable consequences

of the irradiation, if not immediately apparent, will become increasingly so as the months or years progress. If the radiation is delivered postoperatively, an area is treated which has been much modified by surgery; there is residual inflammation, scarring, and, perhaps most important, a markedly impaired blood supply. The surgeon does not wish to have his patient too much disturbed and distressed by irradiation. The radiologist, fearful of producing complications which may place him in a bad light, often indulges in mere token therapy. We are convinced that any man who has control of the radiation therapy and surgery and who is not influenced by any pressures, including economic ones, can evolve no better method of management than the employment of precision preoperative radiation therapy, preferably with supervoltage, followed by radical surgery. Surgeons who have little knowledge of radiation therapy delivered at its best have no more right to judge and evaluate irradiation as a method of management when employed by men with meager experience than they have to judge the capacities of surgery when such surgery is employed by surgeons of meager experience and/or capacity.

Many of the lesions which are treated involving the antrum are not to be classed as radiosensitive lesions. This, however, is not to be regarded as a deterrent to the use of radiation therapy, for we must come to understand that radiosensitivity and radiocurability are by no means synonymous terms. A radiosensitive lesion is far less likely to be cured because of the greater chances of wide local infiltration and widespread metastasis. A lesion which is far less radiosensitive, but still radioresponsive, carries with it a higher chance of radiocurability because of the reduced rate of local spread, less tendency toward deep infiltrative advances, and the reduced rate and more orderly type of metastasis, and, perhaps just as important, the delayed appearance of metastasis. All of these factors lend themselves to greater chance for cure inasmuch as they offer the therapist more abundant opportunities to achieve cure. The higher dosages are, of course, necessary to effect sterilization, and with higher doses come increased incidence of complications attributable to the irradiation. If the irradiation is given after operation, the dosage cannot safely reach cancer-lethal levels. The high incidence of future complications would make heavy therapy a foolhardy venture.

It is for this very reason that the radiation should be given preoperatively and that the so-called irradiation-condemned volume of tissues should be removed by a radical surgical procedure at the proper time. Even in those cases where complete tumor devitalization is not achieved, there can be little doubt that the intensive radiation delivered has beneficial effects. The profound radiation effect on the tumor renders the residual cells qualitatively and quantitatively less likely to set up new foci of malignancy if spillage of cells takes place during the operation and makes less likely the possibility of metastasis. Our experience of the past 10 years indicates that intensive preoperative irradiation to the upper jaw, nasal cavity, ethmoid area, orbit, and neck, followed by radical surgery, is a completely feasible procedure even when the dose is delivered to amazingly high levels.[2] The recognition of the compatibility of intensive precision radiation therapy, as a preliminary to radical surgery of the upper jaw, may bring within the scope of radical surgery at least a few cases now considered inoperable.

Irradiation Technique

Although we do not have the space to go deeply into the problem of technique, it should be emphasized that the peripheral limits of the irradiated volume should be sharply controlled. Day after day during the treatment cycle the treatment should be duplicated precisely. We have been able to deliver radiation in so precise a manner that a sharp line of ulcerative mucositis

from the irradiation develops along the palate, and excision carried out later 2-3 mm. outside of this irradiated area is followed by perfect healing. Our skin flaps have healed perfectly in nearly all cases. We have had no difficulty with skin grafts. Our only problems have been mild crusting in the ethmoid region and osteonecrosis of the pterygoids to a moderate degree. Our doses have varied between 5000 and 11,000 r as delivered on the skin. Our average dose has been around 7500 r delivered in a period of about six to seven weeks. The interval after completion of irradiation to the time of surgery averaged about six and a half weeks.

In cases where metastasis presented in the neck, the metastastic lesion or lesions were treated with relatively small portals along the lines we have worked out in the treatment of cervical metastasis associated with oral malignancy.[4] The objective in the treatment of the cervical metastasis is not to sterilize the disease, but rather to hold the metastasis in check while delivering the radiation to the maxilla. At a later date a cervical node dissection is done if the primary disease is controlled.

Earlier Diagnosis

As is true with malignancy in other sites, antral malignancy far too often is at an advanced state when decisive curative therapy is started.[5,6] In our experience of the past few years, almost 50% of the patients suffering from malignancy are seen sometime during the course of the disease by otolaryngologists, who then treat the patients for sinus infection. Fifty-two per cent of our patients, suspecting dental trouble, were seen by dentists. A relatively high percentage of patients with antral malignancy relate a history of previous chronic sinusitis. It is understandable, therefore, that the symptoms of antral malignancy are mistakenly thought to be related to sinus infection.

The relative infrequency of antral malignancy makes screening of this disease a rather difficult problem, but a technique for the study of desquamating neoplastic cells can be worked out. More frequently exploration of the antrum and biopsy should be carried out. In our experience an antral window is easily made via the oral cavity; it always heals and allows good visualization of the entire antrum. A negative report is no cause for remorse.

Careful Preoperative Work-Up

All patients who are being considered for radical surgery are subjected to a careful preoperative work-up. The patient is brought to the operating table at the maximum fitness for the procedure. Particular attention is given to the improvement of infection in the operative site; this includes the nasal cavity, the oral pharynx, and the skin. Radiation reaction on the skin must be healed and in good condition. The patient's blood picture is made satisfactory by means of preoperative blood transfusions. At least six pints of blood are available at the time of operation.

The patient's confidence, as much as is possible, is won before the operation. The procedure is explained to him and cases of a similar type are often demonstrated to the patient so that he can be put at rest mentally and emotionally as much as possible. We have found that this is an important part of the preparation of the patient emotionally for this radical procedure.

Technique of Performance of the Operation

The tracheotomy is the first procedure, and after proper preparation of the entire head, neck, and shoulder area for the complete procedure, the patient is draped for tracheotomy.

Under local anesthesia a midline incision is made and a routine mid- or high-position tracheotomy is performed between the third and the first tracheal ring. A small square of cartilage is removed. As the opening is made into the trachea, a few drops of cocaine are instilled and thiopental sodium

anesthesia is started. The tape on either side of the tracheotomy tube is sutured to the skin, No. 6 silk being used. A sterile tube carrying oxygen is introduced into the tracheotomy tube.

Ligation of the External Carotid Artery.—An incision about 6 cm. in length is made along the anterior margin of the sternocleidomastoid muscle with the midportion of the incision at about the level of the upper border of the thyroid cartilage.

After mobilization of the bifurcation area of the common carotid artery, the bifurcation area is touched with 10% cocaine solution. The external carotid artery is mobilized and ligated above the superior thyroid artery.

The wound is then closed in layers, a small rubber dam drain being used. The wound skin edges are approximated with interrupted silk mattress sutures.

Ligation of Contralateral External Maxillary Artery.—After the facial notch at the lower border of the mandible is located, a small incision about 2 cm. in length or less is made. The vessels are mobilized and delivered. The artery and the veins are then ligated and the wound closed in layers.

Draping.—After this procedure, the wounds are temporarily dressed, and a drape is placed so as to separate the wounds and the tracheotomy from the upper operative field. The drapes are all sutured to the skin. The anesthetist has complete access to the tracheotomy for purposes of suction, etc. The drapes completely separate him from the field.

Suturing of Eyelids.—The eyelids are carefully sutured with silk sutures. The sutures are left in place at least three weeks (preferably longer) to allow time for cicatrization and contraction, which helps prevent eye-drop and consequent double vision. (See under heading "Temporal Muscle Sling," etc.)

Preparation of Oral Cavity.—The oral cavity and the nasal cavity, which have been previously cleansed with tincture of benzalkonium (Zephiran), are again prepared with the same solution, special care being given to the teeth, which have previously been scaled. The pharynx is packed with moist gauze, and the anesthetist is so advised.

Incision for Maxillary Resection.—The incision is started at the midline of the upper lip, the original Ferguson incision being used, with the exception that the upper limit of the incision stops at least 1 cm. below the inner canthus level of the eye. The wound at this point curves laterally only for about 0.5 cm. The lateral horizontal wing of the incision is not used as a routine. (See under next heading.)

The incision is carried through the entire thickness of the lip, and the skin flap over the face is dissected relatively thin. The periosteum must be left to be removed with the specimen.

The lower limit of the lateral intraoral buccal surface incision is variable in position, depending on the extent of involvement. The line of incision generally is approximately the sulcus area at junction with the gingiva. This incision is extended posterior to and around the tuberosity of the maxilla. As the flap is developed, bleeding is controlled.

Sectioning the Maxilla.—After complete mobilization of the lateral face flap, arrangements are made for the sectioning of the bone. The point of section along the palate is determined by the extent of the tumor. If there is obvious neoplasm involving the nasal cavity, the entire nasal floor of that side, including the septum, is removed oustide the area of irradiation. The line of resection, therefore, is along the edge of the septum of the opposite side. In such a case the upper incisor tooth at the point of incision is removed, the mucosa is incised with a knife, and by the use of a narrow-blade Strycker oscillating saw the bone cut is carried at least half-way posteriorly through the hard palate. The rest of the hard palate is split with a fine chisel. Bleeding here is controlled by packing gauze into the freshly made wound. This gauze

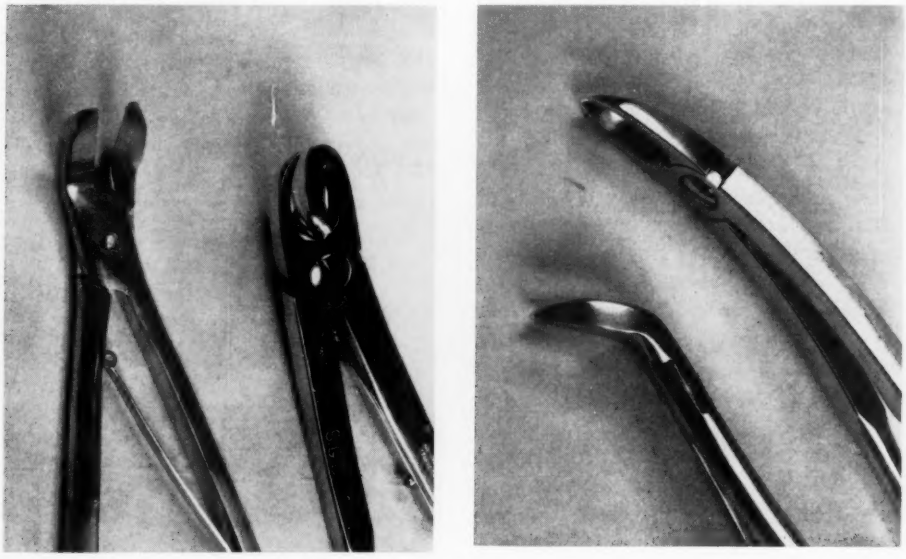
is introduced by firm pressure with a periosteal elevator or chisel. Occasionally it is firmly pounded in with a mallet.

The frontal process of the maxillary bone is cut near its articulation with the nasal bone. The bone is sectioned with the Strycker saw. Here again gauze packing is forced into the wound with a chisel to control bleeding.

As previously pointed out, the Ferguson incision employed by us, which ends below the inner canthus of the eye, does not have a lateral extension to the outer canthus such as is so commonly employed. We feel that this lateral extension is not always needed and should be avoided whenever possible. It is an important factor in the production of the ugly edema which develops in the lower lid postoperatively (Fig. 2). The incision is not needed to bring about the sectioning of the zygomatic process. It should be employed only when needed for adequate exposure to perform an adequate operative procedure. The more skillful the surgeon the less frequently such incision will be found to be necessary.

We have found that in many cases an incision only 1 cm. or 1.5 cm. in length is needed near the lateral canthus of the eye and midway between the upper and lower borders of the zygomatic bone. Through this small incision the zygomatic process is completely mobilized and then severed with a Strycker saw. The incision is begun at the orbital margin laterally at about the level of the upper border of the zygoma and it is carried downward and laterally so as to transect the zygomatic process of the maxilla transversely. Bleeding after sectioning of the bone is again controlled by packing with gauze, using the chisel to thrust the gauze firmly into the bone wound.

All the important bony attachments have been severed now, except the pterygomaxillary synostosis. Now the posterior limits of the incision on the palate are identified, and with a scalpel the incision is extended laterally just posterior to the hard palate and is carried laterally to meet the incision on the buccal surface. This incision is carried deeper with dissecting scissors so



A B

Fig. 1.—*A*, frontal view of new bone-cutting forceps in the left portion of photograph. Compare with frontal view of older bone-cutting forceps to the right. *B*, profile view of new and old style bone-cutting forceps used in cutting pterygoid process. Note reverse bevel.

as to free the muscle attachments to the maxilla. The only osseous points of attachment still remaining are the pterygomaxillary synostosis and the floor of the orbit. The pterygomaxillary synostosis is cut by means of a special bone-cutting forceps with sharply angulated reverse bevel cutting edges * (Fig. 1). An ordinary angled and beveled bone-cutting forceps cannot make the cut.

After the pterygoid is cut free of the maxilla, the forceps are opened slightly and, with the instrument used as a retractor, the maxilla is pulled forward slowly but firmly from its bed. Strong bone-holding forceps are seldom necessary. When needed, they are placed on the maxilla in such a way as to avoid breaking into the antrum and exposing the tumor. By cutting the pterygomaxillary synostosis with bone-cutting forceps the shocking procedure of wrenching the maxilla loose by powerful bone-holding forceps is avoided, sparing the patient unnecessary trauma and reducing the likelihood of spillage of cancer cells. Upon delivery of the specimen, the wound is immediately packed with gauze. After several minutes the packing is removed, and the venous bleeding in the depth of the wound is controlled by ligatures. An effort is made to determine if the en masse removal has been grossly adequate.

Surgery of Ethmoid Area.—The next step is examination of the ethmoid area. The ethmoid cells are curetted, various ethmoid curettes being employed, the type varying with the indication of the moment. The soft tissues which are removed from the ethmoid areas can be studied under frozen section. If this tissue is indicative of malignancy, the entire ethmoid labyrinth can be removed en masse, exposing the underlying dura (Fig. 11). In our three cases removal of the ethmoid was performed with use of a chisel. This was done gently in an effort to avoid tearing the underlying dura. In one case, however, we removed not only the ethmoid labyrinth but the underlying dura and a portion of brain as well.

The sharp edges of bone are rongeured away by means of a duck-bill dental rongeur. At this point all the exposed bony edges are gently rasped and smoothed down. The wound is now washed with 1:10,000 solution of mercury bichloride (mercuric chloride) under a forced spray. This technique has been employed for the past 11 years. Care is taken to make certain that none of the solution passes the pharynx. After the irrigation with mercury bichloride, the cavity is washed with water, then thrombin solution, and packed.

Temporal Muscle Sling to Prevent Eye Drop.—In clean cases where the temporal tendon is adequate, a fair thickness is freed, beginning low in the ascending ramus, and dissected cephalically. The freed tendon is then swung under the orbit and anchored medially so as to cradle and support the eye.

Obtaining and Placing the Skin Graft.—The lateral thigh, previously prepared, is made ready to obtain a skin graft. The skin graft is taken with an oscillating Brown electric dermatome. The skin graft is between 0.020 and 0.022 in. in thickness. Originally the skin grafts were taken freehand, later with a Padgett dermatome, and still later with a Reese modification of the Padgett dermatome.

For the past several years we have been using the Brown electric dermatome and have found this to be best suited for our needs in this operation. A piece of cloth is cut to the size of the skin graft and is placed in the operative cavity as a sort of dry run. The cloth is adjusted as though one were adjusting the skin graft and is gradually trimmed to fit the area to be covered. Wedges may have to be removed in at least one place to avoid overlap. The pattern is then removed from the wound and is trimmed so that about 0.5 cm. is removed from its entire periphery. The

* Obtained by special order through Mr. Henry Ford, representative for Paul J. Gilson Surgical Instruments, Philadelphia.

skin graft is then trimmed to duplicate the size and shape of the pattern. The skin graft is then placed in the cavity and sutured about its periphery with 000 interrupted chromic absorbable sutures (Fig. 12). Some modification of the skin graft may still be necessary despite its pre-preparation from the pattern. As the skin graft is sutured into position, it must be frequently tested by packing to make certain that proper tension on the skin graft will eventually be present when it is completely sutured, and a stent of packing is placed in the cavity.

When the skin graft is completely sutured into position, we are now ready to close the skin flap. The flap is closed by the placement of interrupted upside-down 00 chromic absorbable sutures, the sutures being placed in such a way that the knots are tied on the deep side of the suture. This technique reduces difficulties with wound healing, stitch abscesses, etc.

Special care should be given to muscle approximation in the upper lip. After the buried chromic sutures have been placed, the skin is approximated with interrupted No. 5-0 silk sutures.

Introduction of Gauze Stent.—A most important part of the operation is the packing of the cavity so as to form a stent for the skin graft. We have found that a high-grade type of gauze, such as that used for fluffed-up dressings, makes the best type of packing for this purpose.

Before the packing is placed, however, it is essential that a material be placed over the skin graft which will avoid adherence of the packing to the skin graft. For many years we have been using a perforated cellophane. The film of perforated cellophane is placed in such a way as to avoid as much as possible crinkling and doubling and also to assure complete coverage of the skin graft.

The gauze packing, which usually measures about 12×16 in. is slowly and carefully introduced into the wound, filling it to the level of the remaining palate. It is rather difficult to describe the pressure under which this gauze should be placed. Perhaps the best that can be said is that approximately the same degree of pressure should be exerted against the skin graft as is normally exerted against a skin graft in any situation. To make certain that the packing remains in place, a crocheted netting of heavy cord is sutured to the mucous membrane of the palate along the midline and the free end of the soft palate posteriorly and along the buccal surface below the level of the sutured skin graft laterally (Fig. 13).

Termination of Operative Procedure.—The packing in the pharynx and hypopharynx is removed; the mouth is cleaned with isotonic sodium chloride solution, and the mouth gag is removed. The face is cleaned with water; the silk sutures holding the drapes in position are removed; permanent neck dressings are placed; the tracheotomy tie is made permanent; fluffed-up dressings are placed on the operative side of the face, and a wrap-around dressing is placed, either ordinary gauze or an elastoplast bandage being used. It is our belief that external pressure is necessary to maintain the necessary pressure against the skin graft. It should be noted that we have not had a failure with skin grafts in the past 10 years that they have been used (Fig. 14).

Fig. 2.—Marked edema and ugly contracted funnel-shaped scar of cheek prior to the use of skin grafts in the early 1940's.

Before the patient leaves the operating room, the upper respiratory tract should be completely suctioned to make certain that collected secretions have been entirely removed. The bronchial tree is also aspirated as well as possible with properly adapted catheters. The patient is now ready for the recovery room or for return to the floor, and the postoperative period begins.

The position of the patient in bed is important. It is our belief that the best position is a semireclining one. Because of adequate blood replacement during the operation, the patients return to bed in excellent condition. Shock is never a problem. Important as the care in the recovery room may be, it is vital that a trained team of nurses be in attendance for the postoperative course. Proper oral hygiene is fundamentally important. Proper aspiration of the bronchial tree and care of the tracheotomy wound also are important. Recognition of changes from the normal in the postoperative course is vital to the patient's safety, and a trained team is of inestimable value.

Antibiotics are freely used; the type is somewhat variable. Penicillin and streptomycin in the past have been used in combination. For patients allergic to penicillin, streptomycin and tetracycline (Achromycin) have been used, in other instances streptomycin and chlortetracycline (Aureomycin).

Fig. 3—Operative photograph of resection of the maxilla combined with resection of orbital contents: (1) retracted cheek flap; (2) remaining orbital roof; (3) nose retracted to right; (4) remaining soft palate; (5) large cavity remaining after en masse removal of specimen.

Fig. 4—Operative specimen in radical resection of the maxilla in continuity with the orbit.

Infection has never been a problem. Healing of the skin edges has always been excellent even in cases where high doses of radiation have been employed.

Variations from the Classical Operation

There are several variations to the so-called standard operative procedure described above:

1. *Radical Resection of the Maxilla with Associated Resection of the Orbit.*—When it has been established preoperatively that the orbit should be removed as a part of a properly conceived operation for the cure of the patient, the procedure should be carefully planned so that the orbit and the orbital contents can be removed in continuity with the main specimen (Figs. 3 and 4).

Although it is frequently stated that proptosis can be due to inflammation, it is our feeling that in every case where evidence exists of bony involvement in the region of the infraorbital rim with associated proptosis, or if there is evidence of ethmoid involvement with proptosis, if operation is planned at all, it should be planned with the intent to remove the orbit in continuity.

2. *Block Resection of the Ethmoid Labyrinth Associated with Maxillary Resection.*—It is now possible to state that the ethmoid labyrinth can be removed entirely by incisional surgery down to the dura, and in some cases including the dura, with success. The removal is generally performed

after the resection of the main specimen, including the maxilla and the orbit. It may be that in the future further refinement of technique may make possible the removal of all these structures in continuity. One of us (S. G. C.) has performed three such cases. In one case dura and brain tissue were removed. In this instance the skin graft was placed over the brain itself with success. Because the removed specimen was positive, over 4,000 r was given postoperatively despite a heavy preoperative dose. All three patients are alive from one to three years postoperatively.

3. *Resection of Skin of Cheek Associated with Maxillary Resection.*—In certain instances where there is involvement of the skin or subcutaneous tissue, sacrifice of the entire thickness of the cheek along with the underlying maxilla is necessary. Employing preoperative supervoltage radiation directed to the tissues later to be removed, one can, during the period of radiation therapy, plan a pedicle and flap with a skin lining on the chest. By the time the reaction from supervoltage radiation is complete, if the patient is deemed operable at that time, a radical resection of the upper jaw, including the entire thickness of the face, can be done and immediately repaired with the previously prepared line flap (Figs. 5 and 6).

4. *Resection of Nose Associated With Maxillary Resection.*—In other cases it may be necessary to resect a good portion of the nose. In these instances it is wiser to perform the maxillary resection and to make only a partial closure, leaving the defect resulting from the nasal resection unrepaired. Repair can be made at a later date with more assurance of success by employing an Indian type forehead flap (Figs. 7 and 8).

5. *Partial Resection of the Antrum.*—Partial resection of the antrum is sometimes indicated, although in our judgment it is never indicated when the disease is primary in the antrum. Indications for subtotal resection of the superior maxilla, however, exist.

(*a*) Resection of the palate and alveolar ridge, including the floor of the antrum, for cases of epidermoid carcinoma involving the mucous membrane with extension or bony invasion.

Fig. 5—Operative photograph, resection of maxilla and full thickness of cheek: (1) nasal septum; (2) remaining palate; (3) tongue; (4) pterygoid musculature.

Fig. 6—Postoperative photograph showing plastic repair with use of tubed pedicle flap following resection of maxilla and soft tissues of cheek in continuity.

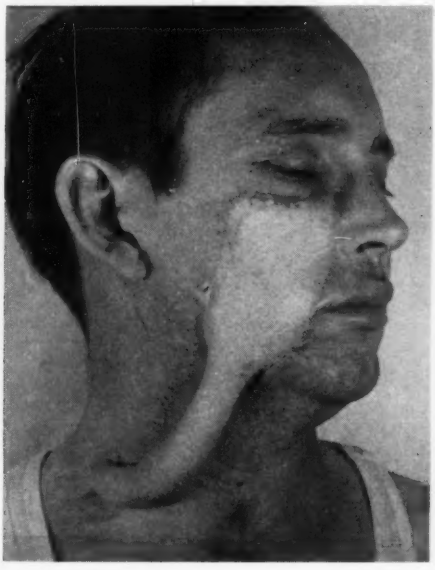


Fig. 7—Operative photograph, resection of nose combined with maxillary resection: (1) retracted remaining left side of nose; (2) large right cheek flap; (3) operative cavity; (4) remaining soft palate.

(*b*) Partial resection of the maxilla, justified in tumors which have originated in the nasal cavity and which have relatively limited extent. In general, these lesions are treated too conservatively.

(*c*) Invasive epidermal tumors involving the periorbital structures, bony floor of the orbit, and extension to the ethmoid area. In such cases, radical resection of the orbit and surrounding tissues, the roof of the antrum and floor of the orbit, anterior wall of the antrum and ethmoid, are resected with a large flap previously prepared on the forehead employed for closure (Fig. 9).

(*d*) Resection of the mandible, floor of the mouth, partial glossectomy, tonsillectomy, resection of soft palate and palate in cases of epidermoid carcinoma, squamous-cell type, involving the

Fig. 8—Postoperative photograph during staged plastic repair of nasal defect employing Indian-type forehead flap following resection of maxilla and nose in continuity.

Fig. 9.—Operative photograph, resection of soft tissues of cheek, orbital contents, ethmoid and frontal sinuses, and superior portion of maxilla in continuity: (1) operative defect; (2) donor site of scalp flap covered with split-thickness skin graft; (3) scalp flap previously lined with split graft to form new nasal surface.

Fig. 10—Postoperative photograph following resection in a case of epidermoid carcinoma, squamous-cell type, involving the retromolar area with extension to involve the soft palate and palate.

Fig. 11.—Special view showing depth of resection and total ethmoid ablation with skin graft placed over dura or brain.

retromolar area with extension to involve the soft palate and palate (Fig. 10).

(*e*) Recurrent ameloblastoma originating in the mandible involving the deep pterygomaxillary space or mixed tumor in this space. In such cases, we resect the zygoma entirely and sweep down to resect the posterior wall of the maxilla and the remaining ascending ramus of the mandible.

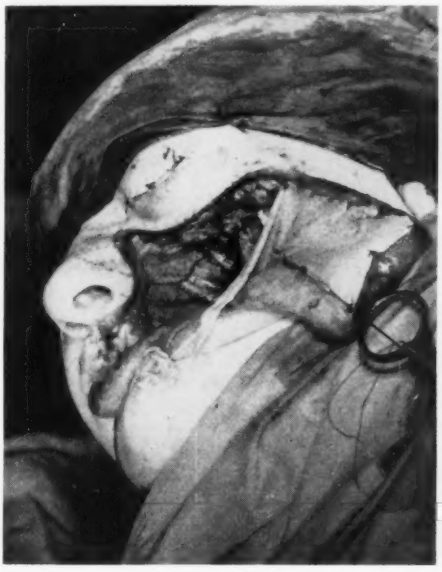
(*f*) Electrocautery of antrum is reserved for cases of recurrence only.

Summary of Experience

The views expressed in this paper are based on an experience of 132 cases of lesions involving various structures of the maxilla, including 65 lesions considered to be primary in the maxillary antrum.

Since the technique herein described of intensive preoperative radiation therapy has been employed, 15 patients have had an opportunity to survive over five years. In this group a survival rate without evidence of disease at the end of five years of 47% was achieved. Fifty-seven per cent of our patients have been alive and well for periods varying from two years up. In the group, 23 patients were rejected for surgery; 3 received no treatment of any kind because of the far-advanced nature of the disease. The patients rejected for surgery were treated with radiation only, and one has survived five years.

Fig. 12.—Skin graft being sutured in place in the cheek flap and remaining operative cavity.



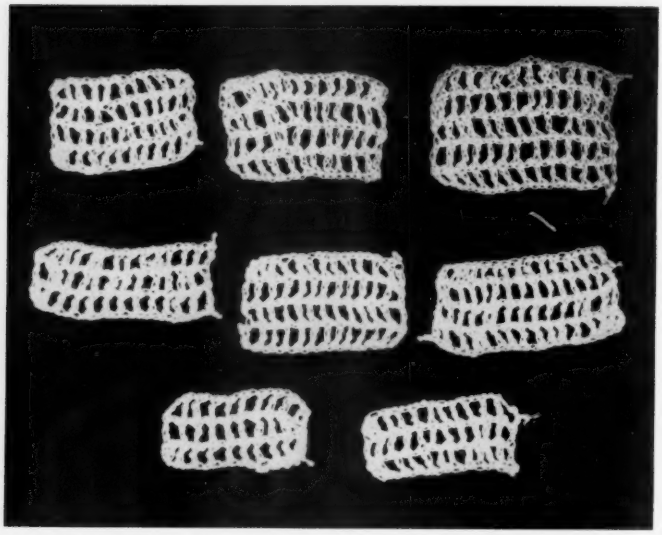


Fig. 13.—Example of crocheted "hammock" of different sizes made of heavy upholstering cord. The hammock is sutured to the mucous membrane of the palate and the free edge of the soft palate and along the buccal surface to hold the gauge stent in position.

Fig. 14.—Ideal result. Photo taken in April, 1952, four years postoperative. Patient had resection of upper jaw for twice recurrent fibrosarcoma. This shows remarkable cosmetic result, which can occasionally be achieved. This case, as have all cases for the past 10 years or more, had a skin graft.

Fig. 15.—A case of radical resection of upper maxilla, radical resection of orbit and total ablation of ethmoid area with skin graft. *A* represents skin graft over resected ethmoid area.

Conclusion

We feel that definite progress in the management of malignant antral disease will attend the use of the principles of management described. These principles do not lend themselves to summary since they must be viewed against the background of past ex-

perience to be fully appreciated. The details and refinements of operative technique are described.

Workers in the field of antral malignancy are from diverse disciplines and are still far from unity of thought regarding the early diagnosis and selection of the best method of management for this disease. We feel that the lesion is in the province of the otolaryngologist and that the proper management is a combination of precisely controlled preoperative irradiation followed by surgical removal of the condemned tissues.

This method is best carried out by one person thoroughly familiar with the disease and the radiologic and surgical principles involved.

3234 Powelton Ave. (4).

REFERENCES

1. Ackerman, L. V., and del Regato, J. A.: Cancer: Diagnosis, Treatment, and Prognosis Ed. 2, St. Louis, The C. V. Mosby Company, 1954, pp. 198-227.

2. Castigliano, S. G.: The Role of the Otolaryngologist and Ophthalmologist in the Management of Tumors of the Head and Neck, Tr. 39th Ann. Meeting Indiana Acad. Ophth. & Otolaryng., 1956.

3. Castigliano, S. G.: Inoperability versus Incurability in Tumor Management, Pennsylvania M. J. 52:1364-1370, 1949.

4. Castigliano, S. G., and Rominger, C. J.: Dissection of the Neck After Intensive Irradiation to the Neck, Am. J. Roentgenol. 69:771-788, 1953.

5. Castigliano, S. G., and Rominger, C. J.: Distant Metastasis from Carcinoma of the Oral Cavity, Am. J. Roentgenol. 71:997-1006, 1954.

6. Castigliano, S. G., and Rominger, C. J.: Metastatic Malignancy of the Jaws, Am. J. Surg. 87:496-507, 1954.

7. Devine, K. D.; Scanlon, P. W., and Figi, F. A.: Malignant Tumors of the Nose and Paranasal Sinuses, J. A. M. A. 163:617-621, 1957.

8. Mattick, W. L., and Streuter, M. A.: Carcinoma of the Maxillary Antrum, Surgery 35:236-242, 1954.

9. Öhngren, L. G.: Malignant Tumors of Maxillo-Ethmoidal Region: Clinical Study with Special Reference to Treatment with Electrosurgery and Irradiation, Acta otolaryng., Supp. 19, pp. 1-476, 1933.

10. Schall, L. A.: Malignant Tumors of the Nose and Nasal Accessory Sinuses, J. A. M. A. 137:1273-1276, 1948.

11. Ward, G. E., and Hendrick, J. W.: Diagnosis and Treatment of Tumors of the Head and Neck, Baltimore, Williams & Wilkins Company, 1950, pp. 465-490.

12. Windeyer, B. W.: Malignant Tumors of the Upper Jaw, Brit. J. Radiol. 16:362, 1943; 17:18, 1944.

Pharyngolaryngeal Disturbances Due to Cervical Spondylosis

A. LASKIEWICZ, M.D., London

In 1898 Pierre Marie and in 1908 Mare Hardour drew attention for the first time to disturbances of the throat and esophagus in the course of rheumatic inflammation of the cervical spine. J. Terracol in 1925, Halphen in 1926, J. Piquet in 1951, and Boulet and Gerecourt in 1952 have described precisely these changes as "syndrome pharyngo-oesophagien" in the form of motor disturbances of the pharynx and the upper part of the esophagus, as well as the feeling of sorness and stiffness there. This syndrome due to rheumatic changes mainly in the middle part of the cervical spine was finally defined by these authors as "dyskinesis pharyngo-oesophagiennes" or "arthrose cervicale dysphagique" (Terracol). This author distinguishes cases in which motor disturbances of the pharynx and the upper part of the esophagus prevail, causing difficulties in swallowing, from the second sensitive form called also "paraesthesiae pharyngis." This type of dysphagia is characterized by a unilateral feeling of soreness and tenderness of the neck, radiating toward the cornu major of the hyoid bone, posterior margin of the thyroid cartilage, or below the cricoid cartilage. In the latter case a feeling of oppression, drawing, and pricking in the throat, as well as tenderness in the laryngeal region, is prominent in the clinical symptomatology and is aggravated on empty swallowing. Vernet (1936) believes that insufficiency of the upper pharyngeal constrictor often occurs in affections of the glossopharyngeal nerve while the anterior pharyngeal wall pushes forward like a curtain (*movements de rideau*). Brunner pointed out that tonus relaxation of the lower pharyngeal constrictor, besides a strong contraction of the cricopharyngeal muscle, often occurs in these cases. He observed roentgenologically in most cases striking concretions, exostoses, and syndesmophytes around the articulation in the middle portion of the cervical spine. In only a few cases he established a diagnosis of acute inflammatory process of the cervical spine in which the symptoms from shoulder, arm, and hand prevailed as parts connected closely with the innervation of the seventh cervical segment. Precise radiography of the small cervical joints, taken of the neck in the exact lateral projection according to the method of Grashey, Nievergelt, and Doerffel shows arthritic-deforming changes within the third to sixth cervical vertebrae, likewise as syndesmophytes, narrowing the intervertebral foramina of the third to seventh cervical segment. A. Seiffert cites a case in which exostosis of the middle part of the sixth cervical vertebra narrowed the esophageal passage. Jaffe had cited a case of spondylitis ankylopoetica in which analogous disturbances from the gullet were produced by its flattening and moving aside. Widespread forms of spondylitis cervicalis deformans with pharyngoesophageal disturbances were described by A. Thost, R. Fernandez, C. Wiethe, A. Schlachter, and P. Falk. There were mainly bone protuberances of the margins of the third, fourth, sixth, and seventh cervical vertebrae in which neuralgic pains of the cervical-pharyngeal plexus, radiating to the shoulder and the upper extremity were prominent in the clinical picture, besides difficulty in swallowing solid food particles. In the case of C. Wiethe we were dealing, moreover, with unilateral palsy of the palate as a conse-

Accepted for publication March 8, 1957.

quence of post-traumatic periosteal hyperplastic changes of the second and third cervical vertebrae. As for pathogenesis and morphology of the changes in question, it must be emphasized that the process involves at first the small articulations of the cervical spine (degenerative joint disease [R. Jackson]), from which it spreads into the vertebral periosteum as a bone exostosis of its anterior surface in different form and dimensions. Around the intervertebral foramina it causes narrowing of these foramina.

Diffuse proliferative periosteal thickening of the anterior surface of the cervical spine was described by Schmorl and Rokitansky as exostoses resembling sugar icing *(Zuckergussartige Knochenwucherung)* of the vertebra and its ligamentum latum. In some cases the osteophytes of the lower border of the cervical spine form roofing-tile shapes overlapping partially. In protracted cases these neighboring formations sometimes grow together. Often also the posterior part of the cervical spine is not spared from this complication, while the anterior part of this spine becomes deformate and arched forward to the front (ankylokyphosis cervicalis). Beneke supposes that this process starts with diminishing of elasticity of the intervertebral ligaments and disk as a symptom of aging with consequent loss of elastic resistance to shock, affecting the cervical spine in its long axis. Excessive strain of these ligaments and the surfaces of the spinal articulations may be caused by sudden emotional turns of the head aside or backward, work in an inclined position (especially in surroundings with increased pressure, for instance, in caissons), or prolonged toil with the hands extended upward, as among upholsterers, decorators, and typists. Unnatural hyperextended, slight spasmotic holding of the head and the neck in a sitting position as well as in a standing one is responsible for degenerative changes within the articulations of the cervical spine (spondylosis cervicalis). This prolonged traumatic action causes relaxation of the

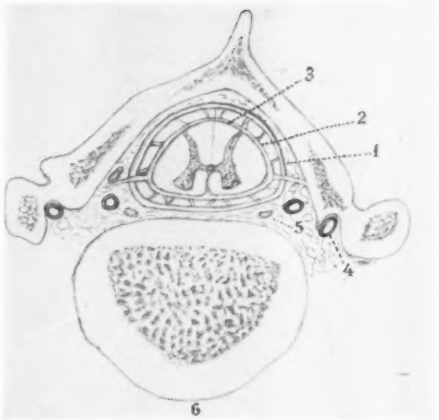


Fig. 1.—Transverse section through the upper cervical spine on the level of the third cervical vertebra; (*1*) dura sheath; (*2*) pia mater; (*3*) spinal cord; (*4*) spinal artery; (*5*) vertebral venous plexus; (*6*) corpus vertebrae.

ligaments and articular bands, which easily become edematous, as well as of the meningeal sheaths surrounding the lateral spinal roots (Janzen, Zeidler, Kuhlendahl, Junghans). During sudden backward movements of the head there occurs also dislocation of the vertebral artery, which presses suddenly on the sympathetic plexus. Any irritation of the neural spinal roots is connected with reflex irritation of the cervical sympathetic system; consequently the eye and the inner-ear irritations are prominent in the clinical features. According to the comparative statistics given by E. Schindel 44.5% of heavy physical workers and 34.5% of mental workers and clerks show these changes within the cervical spine. A small difference amounting to only 10% proves that the petty prolonged traumatic injuries (microtrauma or "Peitscherschnurtrauma," according to Bourmer) of the spinal articulations and ligaments come into consideration. This part of the cervical spine is plentifully innervated by the posterior branches of the spinal nerve roots (Luschka, Rüdinger, Valentin). The new bony formation of the cervical vertebral margins must be considered as a consequence of prolonged irritation of the spinal articulation apparatus, there causing a considerable limitation of its

mobility (*funktionelle Knochenneubildung*) and also often its deformity. Moreover, the intervertebral ligaments, deprived of their elasticity, can easily bend sideways, pressing upon the intervertebral nerves. Compression of constricted intevertebral foramina, sometimes by osteophytes growing there upon the spinal roots, their dislocation produced by traction on dentate ligaments, and adherence of the ligamenta flava to the dura, are responsible for the neuralgic pains when the neck is extended. Wide adhesions within the dura aggravate these factors by compression of the anterior spinal artery. Histological examination of exostoses and the roofing-tile osteophytes of periosteal origin shows a parabolic inlay of the bone plates (lamellae) as a consequence of permanent pressure upon the long axis of the cervical spine. (Fig. 2). Especially within the osteophytes these bone plates are situated horizontally to the direction of the acting force, looking like a parabolic compound curve. In early stages of spondylitis deformans we find on the points of this ossification fibrous cartilage in which the ossification centers are situated in arched form in relation to the external surface of the cervical spine, forming the bend of a parabolic line directed downward. In severer cases we can find this process even in the posterior parts of the spine. One of the important factors of spondyloarthritis cervicalis deformans, according to Roessle and Fischer, is the spreading of infection from the dental-periapical-tonsillar foci toward the submaxillary neck glands and throughout the perivascular-perineural spaces to the roots of the different intervertebral cervical nerves, causing typical neuralgic pains of the cervical plexus. Simultaneously the ligaments of the adhering cervical muscles and the intervertebral articulations are affected on the same underlying basis (tenonitis, capsulitis, arthritis intervertebralis). In the course of time deposition of calcium takes place, with subsequent ankylosis of these joints (spondyloarthritis ankylopoetica). The influence of endocrine glands (parathyroids, anterior pituitary, gonads) is probably indirectly concerned in the etiology of the disease in question, controlling the vasomotor state and the behavior of the spinal vessels. Its frequent constriction, even on the underlying allergic basis, remains not without influence upon the physical conditions of the cervical vertebral bone and its ligaments; likewise the advanced disturbance in purine metabolism is associated with frequent attacks of acute and chronic spondyloarthritis. Clinical manifestations of these advanced changes were defined before as the syndrome of Bechterew, P. Marie, or Strümpell, while the advances in radiography of the neck have explained their real etiology. Exostoses of the 8th, 9th, and 10th dorsal vertebrae in the course of spondylitis deformans can narrow partially the esophageal lumen at the level of the diaphragm, as in cases described by Zahn and Pineson.

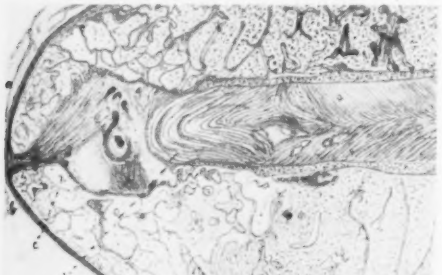


Fig. 2.—Longitudinal section through the osteophytes of the lower border of the fifth cervical vertebra (*a*) and the upper one of the six cervical vertebrae (*b*) growing together. Parabolic inlay of the bone plates visible (*c*); intervertebral disk (*d*) (a man of 69 with spondyloarthritis cervicalis deformans, died from coronary thrombosis).

Symptomatology of the clinical manifestations in cervical spondylosis should be divided into (*a*) pharyngeal, (*b*) laryngeal, (*c*) esophageal and (*d*) aural disturbances and more distant neuralgic pains. The feeling of burning, stiffness, and tenderness at the level of the larynx, sliding of which upon a bone protuberance of the anterior vertebral surface is sometimes experienced by these patients during swallowing, is often connected with prickly pain radiating to the

ears and occipital region. Burning of the posterior part of the tongue (papillae circumvallae region) interrupted or continuous (glossodynia) is a consequence of irritation of the glossopharyngeal nerve. Engman has defined this symptom as the feeling of a burning wound on the tongue and the pharynx. Soreness and lump in the throat (globus) call forth a constant urge to clear it and cough with expectoration of a small quantity of mucous secretion, which together with the said paresthesias, is responsible for so-called frequent empty swallowing, "aerophagia" of varying intensity. From the upper part of the esophagus we note some difficulties in swallowing of solid food particles mainly produced by insufficiency of the constrictor pharyngis medius and inferior rather than by mechanical obstruction due to the bone protuberances of the anterior part of fifth to seventh cervical vertebrae. In laryngoscopy the remnants of saliva in both piriform valleculae was striking beside an atrophic pharyngeal mucosa, drying here and there as if it were varnished (Fig. 3). This part of the pharyngeal mucosa is characterized by considerable decline of reflexes, so the patient is easily able to probe his pharynx and larynx like a phantom. Any act of swallowing is connected with pricking pains radiating toward the hyoid bone and below the cricoid cartilage. The larynx is not uninvolved in the entity of these syndromes, showing usually a pallid color of its mucosa as a consequence of continued irritation of the cervical sympathetic branches and ganglia. A small quantity of secretion covers the vocal cords, revealing insufficiency of the musculus vocalis during phonation, undoubtedly on the same basis. Phonasthenic disturbances are in these instances more frequent, explaining the symptom of vocal weariness and hoarseness during prolonged speech as it was described by Heck and by Tarneaud under the designation "syndrome sympatico-vocal." More distressing symptoms of dysphagia from the upper part of the esophagus are evident in cases of large exostoses of the sixth to seventh cervical vertebrae or bone spans joining them. In the last event besides the pharyngolaryngeal symptoms mentioned we were faced with neuralgic pains of varying intensity, radiating to the neck, occiput, and top of the head. Disturbances of the inner ear, such as difficulty in hearing speech and whisper voice, middle and high tones, as well as giddiness with bilateral horizontal nystagmus in three cases of arthritis cervicalis of the fifth to seventh cervical vertebrae, were described by Barré in 1926. Maspetiol, Chardin, and Millard distinguished the following disturbances in the inner ear in the course of cervical spondylosis: (1) a cochleovestibular, (2) a pure vestibular, and (3) a cochlear variety. In the first giddiness prevails over hardness of hearing of a mixed type, conductive-perceptive; Romberg's sign is positive. In the second form giddiness with horizontal-rotatory nystagmus (on both sides) is prominent as a clinical feature. Positional nystagmus is also easily elicited. Sometimes dysharmony of vestibular symptoms is evident in these cases (Mayoux). Finally, in the third variety, unilateral hardness of hearing of the perceptive type occurs without any vestibular symptoms. Barré explains this symptom as a constant irritation of the sympathetic plexus around the vertebral artery, and simultaneously venostasis within the spinal veins is responsible for

Fig. 3.—Drawing of laryngoscopic view from a case of cervical spondylosis, showing remnants of mucous secretion and saliva in both piriform valleculae.

intensity of neuralgic pains numbered among the so-called remote symptoms of spondylitis cervicalis deformans. Fine branches of this periarterial sympathetic plexus can be irritated in their course through the intervertebral foramina to the cervical sympathetic by the spinal periarticular process. These fine connections of both cervical sympathetic groups were first described by Luschka (then called rami sympathetici recurrentes Luschka), whose irritation is often transmitted to the motor cells of the given spinal segment resulting in one-sided contraction of the cervical muscles. Neuralgic pains in the course of spondylitis appear as occipital, top-of-the-head, temporal, neck, and shoulder-region pains, radiating to the arms and hands. The first kind of paresthesia is to be explained as pressure upon the greater occipital nerve (n. Arnoldi), which is formed by junction of the posterior roots of the cervical nerves C-2 and C-3. These pains of prickly character are accompanied by a feeling of tension in the muscles of the neck during movements of the head, which together with the ear symptoms are defined as posterior cervical syndrome (Barré). Involvement of the cervical vertebrae, C-4 and C-5 is connected with prickly and stretching pains of the inferior part of the neck and upper part of the chest radiating to the external surface of the shoulder and arms, those of C-6 and C-7 with pains in the posteriosuperior part of the chest and externolateral part of the arms, radiating to the first, second, and third fingers, as well as to the external part of the palm. But involvement of C-8 is responsible for prickly pains of the internal surface of the arms, palms, fourth and fifth fingers, which symptoms were defined by Berschauer as "Lasègue of the upper extremities." In the last condition there occurs more frequently complete palsy of the shoulder-arm plexus, as was pointed out by Léri. Psychical disturbances in the course of the named disease of the cervical spine seem to have their expression in the described oropharyngeal paresthesias, connected always with neuralgic pains of the head, the neck, the chest, and arms, accounting for the hypersensitivity of these patients. Often the patients are in continual fear that they are affected by neoplasm of the tongue or the throat, or by angina pectoris, and that their complaints are incurable. Anginoid sensations were caused by aerophagia, i. e., pressure of the swallowed air in the stomach upon the diaphragm. The diagnosis of different cases is based at first on precise anamnesis and laryngoendoscopical examination of the hypopharynx and esophagus in order to exclude neoplasm, which sometimes starts insidiously from this part of the digestive tract. Patients' statements are usually full of details of their different complaints and their ostensible causes which are often in contrast to the results of our examination. Then radiography of the neck in Grashey or Doerffells projection frequently reveals the real cause of the disease, confirmed also by sensitivity of the spinal roots and the cervical plexus during palpation as well as crepitation during movements of the head in all directions.

Among 12 cases of acute and chronic arthritis of the cervical spine observed by me during the period from 1943 to 1957 there were 11 men and one woman, 23-74 years of age, in whom disturbances of the inner ear predominated in seven cases, while in the remaining five cases there were pharyngolaryngeal symptoms allied to remote neuralgic pains. The first group includes seven men 23-67 years of age, workers by profession, who could be divided in two subdivisions, i. e., four cases of acute arthritis of the cervical spine in men of the ages of 23, 27, 30, and 35, in whom the arthritic process, involving also the knee and spring joints, was connected with recurrent chronic tonsillitis. The following three cases of a more protracted course of changes in the cervical spine were related to three men, aged 56, 67, and 45, the first of whom, a carpenter, showed the following complex of inner ear symptoms: bilateral hardness of hearing to whisper voice ¾ m. shorten-

ing of the lower and medial tones; Schwabach and Rinne tests positive; ear noises of higher intensity; giddiness during side movements of the head. Caloric test on the right side shortened; rotatory, unchanged; Romberg to the left side; lack of spontaneous nystagmus. There was painful pressure on the cervical spine from the fourth vertebra downward as well as during movements of the head on both sides with distinct crepitation within the spine articulations. Moreover, he complained of painful sensation during swallowing at the base of the tongue and at the level of the cricoid cartilage; phonasthenic disturbances in the form of weakness and breaking of the voice; pains in the lower part of the neck, upper part of the chest and shoulder, radiating to the external part of the arms, the hand, and the first and second fingers. Radiography of the neck in Grashey projection revealed changes within the fourth and fifth vertebrae in the form of exostoses of the anterior parts and the lower margins of these spines. Margins of the fifth and sixth vertebrae show small exostoses. Small intervertebral articulations are slightly thickened and of unequal contours. In the second case (a man of 67, locksmith by profession) we were dealing also with hardness of hearing for speech and whisper voice 5/2.5 m. and the lower-medial tones of the audiometric scale; Schwabach and Rinne tests positive; horizontal-rotatory nystagmus to the right side; left-sided caloric test slightly decreased; turning test unchanged. The patient's complaints were aural noises, pains in the neck during turning of the head, especially backward, while crepitation in the cervical spine articulations was experienced, accompanied by short giddiness. Palpation of the spine was painful from the third vertebra downward. Radiography of the neck revealed distinct proliferative exostotic process of the anteroinferior margin from the third vertebra downward in the form of roofing tile overlapping the same exostoses of the upper margin of the next vertebra (Fig. 4). The small spine articulations of unequally thickened contours and

Fig. 4.—A man of 67. Roentgenogram of the neck in Grashey incidence, showing exostoses of the anterior inferior margins of the spine from the third vertebra down.

narrowed intervertebral foramina explain the pain during palpation of the sixth, seventh, and eighth cervical nerves and movements in the spine articulations, felt in the posterosuperior part of the chest and external parts of the arms and hands. As to the throat, pains in the posterior part of the tongue and during swallowing were mentioned. There was hoarseness during prolonged speech, connected with a feeling of a lump, cranberry, or orange seed in the throat. Clinical diagnosis was established as spondyloarthritis cervicalis ankylopoetica subsequente laesione auris interioris vasomotoria. The third case in Group A was a man of 45, a building worker, in whom in the course of the same changes within the cervical spine inner ear (cochlear) symptoms prevailed, with disturbances from the oropharynx and larynx. There was hardness of hearing for speech, whisper voice, low

and middle audiometric tones. Rinne test was positive; there were high ear noises, but the vestibular symptoms were lacking; caloric and turning tests unchanged. There were complaints from the oropharynx, as periodic soreness of the tongue, feeling of pressure and lump in the throat. On laryngoscopy remnants of saliva in the right piriform vallecula. Radiography of the neck showed spondylitic changes limited to the fifth and sixth cervical spine in the form of flat protuberances of the lower margins of the spine; intervertebral foramina narrowed and of unequal contours. In Group B we notice four cases of men 42-60 years old and one woman of 74. In the first case we were dealing with a man of 42 who complained of sore throat, feeling of tightness, and a lump, calling for constant clearing of the throat and empty swallowing. During prolonged speech distinct symptoms of weakness of the voice were evident. Radiography revealed hooked bone protuberances of the lower margins of the fifth, sixth, and seventh vertebrae, the distances between which had become markedly reduced; intervertebral foramina of unequal contours. The second case was a man of 48, a farmer by profession, who experienced soreness, feeling of a lump of orange seed in the throat behind the larynx, aggravated during swallowing, as well as hoarsness during prolonged speech. Simultaneously he was troubled by feelings of stiffness of the neck and neuralgic pains, radiating to the top of the head. Radiography showed small exostoses on the lower margins of the fifth and sixth cervical vertebrae, with narrowed margins of the intervertebral foramina. Laryngoscopically, remnants of saliva were seen in the right piriform vallecula; larynx unchanged. The third case was a man of 58, installer, who complained of pains during swallowing, radiating to the posterior part of the tongue and the ears, necessitating constant clearing of the throat, and cough. Laryngoscopically, pharyngeal mucosa in atrophic state as if varnished. The vocal cords do not close tightly during phonation, leaving triangular space in the posterior part of the glottis. Palpation of the lateral laryngeal surface was painful. Roentgenologically, the cervical spine from the third vertebra downward shows wide hooked exostoses of lower margins; intervertebral foramina of unequal contours, narrowed; posterior part of the cricoid cartilage, wide-spreading calcifications. In the fourth case we were dealing with arthritic-rheumatic changes in the hands and the legs in a 60-year-old man who was suffering from soreness, pains, and a lump in the throat, as well as from dysphagia during swallowing, mainly at the laryngeal level. These troubles were often aggravated, causing a feeling of regurgitation. Therefore he was in continual fear of being affected by neoplasm in the throat or gullet. Phonasthenic disturbances in the form of weakness of the voice, and necessity to spit out the mucous secretion. Lateral radiography of the neck shows osteoarthritic changes within the fifth to eighth cervical vertebrae, as well as overlapping bone exostoses of their lower margins, considerably reducing the intervertebral spaces. Thickening and expanding of the intervertebral foramina from the fourth downward. Pharyngeal mucosa in atrophic state, pallid; piriform valleculae filled with saliva and mucous secretion. Endoscopic examination of the hypopharynx and esophagus has not revealed any changes there. Finally the case of a woman of 74, seamstress by profession, complaining of headaches and neuralgic pains in the back and occiput, radiating to the shoulder and hands, especially during the night. Distinct crepitation is perceived while turning the head sideways or backward. There was a feeling of burning in the posterior part of the tongue and the soft palate, as well as tightness and lump in the throat. Hoarseness was periodically evident during prolonged speech. But during swallowing of fluid and half-solid food the patient experienced a disagreeable sliding of the larynx upon a bone protuberance of the anterior vertebral surface. Laryngoscopical examination showed the following: thinness of the pharyngeal mucosa drying up here and there;

lingual tonsil slightly increased; in both piriform valleculae remnants of saliva and mucous secretion; laryngeal mucosa pallid; musculus vocalis slightly insufficient during phonation. Radiography revealed wide bone productive changes within the cervical spine from the third cervical vertebra downward, more intensified on the laryngeal level, where osteophytes of the anterior vertebral surface form roofing-tile shapes, overlapping the neighboring spine (Fig. 5). Rokitansky had defined such a type of changes as spondylitis deformans in the form of sugar-icing-like exostoses of the spine. These bone protuberances are responsible for considerable contraction of the intervertebral foramina and spaces, which become reduced. Therapy of the cervical syndrome and the disturbances of the throat, larynx, and the upper part of esophagus connected with spondyloarthritis cervicalis must be at first conservative and symptomatic. In mild cases immobilization of the neck by means of a plastic cervical collar, lined with chamois (Clarke), additional electrical massage of the posterior part of the neck, and use of friction with iodine oil (Anajol) are usually sufficient; short waves on both sides of the neck, phenobarbital-bromide internally. If the patient's condition does not deteriorate, this treatment may be continued for a further three months, but any worsening or failure to stop the progress in the form of persistent giddiness, tinnitus, or pharyngolaryngeal disturbances demands more energetic procedure. This should consist in paravertebral blocking of the sympathetic and cranial parasympathetic pathways by using a 1% procaine solution. The needle, bent at an angle of 45 degrees to the longitudinal spinal axis, should be carefully introduced into the second, third, and fourth posterior intercostal spaces right down to the lateral process of the given vertebra. When the patient complains of pain radiating along the upper part of the thorax and shoulder 1.5 cc. of 1% procaine is injected to block the nerve pathways involved. In three cases after applying this procedure the outcome was satisfactory, resulting in cessation of giddiness and marked diminution of tinnitus noises and also of pharyngolaryngeal disturbances. It was sufficient to continue the further conservative treatment. G. Passe and R. S. Lewis have stated that blocking of stellate ganglion and superior cervical ganglion, particularly their excision, has a favorable effect in obstinate cases, producing vasodilatation within the middle-inner ear and also in the corresponding nasal cavity. On the basis of the principle that a real cause of the cervical syndrome with pharyngolaryngeal disturbances consists in edema of the intervertebral ligaments and meningeal sheaths surrounding the lateral spinal roots as well as of rami communicantes to the corresponding sympathetic ganglia, J. Piquet recommends deep x-ray irradiation, using 1500-3000 r at intervals of 10-21 days.

Fig. 5.—A woman of 74 (spondylitis deformans). Roentgenogram of the cervical spine taken in Grashey incidence, showing osteophytes of the anterior vertebral surface from the third cervical vertebra downwards.

As for the surgical treatment of cases in which failure to stop the progress of the process is evident, the operation should consist in removal of larger bone or cartilaginous osteophytes limited to the anterior surface of the cervical spine, which press on

the hypopharyngeal or esophageal wall. P. Falk proposes to perform a delicate operation on the hypopharynx and the upper part of the esophagus from the cervical spine, using an incision along the internal border of the left sternocleidomastoid muscle. Then the prominent osteophytes could be easily removed with chisel and cutting bone forceps. Decompression of the roots by division of dentate ligaments and enlargement of constricted intervertebral foramina should be also considered in more advanced cases before the disability becomes too severe. It its worth mentioning that Kashiwada recommends cutting off the top of an enlarged epiglottis which touches a wide part of the base of the tongue; disappearance of the sensation of tightness and a lump in the throat was noted in a great number of cases. Finally psychotherapy could also be applied in most cases, since a frequent occurrence in these patients is a permanent psychological disturbance characterized by cancerphobia. A wise solution of this problem by explaining the real cause of pharyngolaryngeal examination, excluding the presence of neoplasm, is a great consolation for these patients and simultaneously also a stimulating encouragement in their life.

Summary

Cervical spondylosis belongs to the degenerative changes within the small articulations of the cervical spine as a consequence of overstrain by prolonged traumatic action on the surfaces of the spinal articulations, their articular bands and ligaments, which become relaxed. This is caused mainly by sudden emotional turning of the head while working in an inclined position or prolonged toil with hands extended upward. More advanced changes within the cervical spine should be defined as spondyloarthritis cervicalis deformans, the appearance often proceeding from spreading infection from the dental periapical or tonsillar foci throughout the lymphatic system and the perivascular and perineural spaces to the roots of the different intervertebral cervical nerves and small spine articulations. As a consequence there occurs pressure of edematous tissue upon the spinal roots of different cervical sectors, and with time deposition of calcium takes place within the spinal articulations. Then a new bone formation at the lower borders of different cervical vertebrae form roofing-tile shapes, overlapping partially and in advanced cases growing together. There occur a burning sensation of the posterior part of the tongue, soreness, and lump in the throat, producing constant urge to clear it and frequent empty swallowing (aerophagia); difficulty in swallowing of solid food particles (insufficiency of the constrictor pharyngis medius and inferior) associated with prickly pain, radiating to the lower border of the cricoid cartilage and also to the ears and occipital region. In the larynx the mucosa has a pallid color; there is insufficiency of the musculus vocalis, and phonasthenic disturbances often occur in the form of vocal weariness and hoarseness, defined also as "syndrome sympatico-vocal." Among the remote symptoms of spondylitis cervicalis are mentioned neuralgic pains of the occipital, top of the head, temporal, neck, and shoulder region, radiating to the arms and hands. Diagnosis is based on the laryngoendoscopic examinations and radiography of the neck, palpation of the spinal roots and crepitation during movements of the head and neck. Among 12 cases of spondyloarthritis cervicalis there were four cases of acute arthritis of the cervical spine in the course of polyarthritis rheumatica and recurrent chronic tonsillitis. In eight cases we were dealing with degenerative changes of the spinal articulations, connected with new bone formation within the 3d to 7th cervical vertebrae as a consequence of prolonged traumatic action; 63% of the described cases are believed to be of microtraumatic origin, in which overstrain of surfaces of the spinal articulations, their articular bands and ligaments was produced by long-lasting hyperextended, slightly spasmodic holding of the head and the neck as well as work in an inclined position. In

all cases the above-described pharyngolaryngeal and esophageal symptoms were clearly evident, but in seven cases disturbances of the inner ear prevailed in the clinical features in the form of hardness of hearing, ear noises, tinnitus, and giddiness. Bilateral horizontal-rotatory nystagmus was aggravated during turning of the head, besides lowering of caloric and unchanged rotatory tests.

Treatment consisted in these cases in paravertebral blocking of the sympathetic and cranial-parasympathetic pathways with use of 1% procaine; immobilization of the neck by means of a plastic cervical collar; additional electric massage of the posterior part of the neck; use of friction with iodine oil; short waves on both sides of the neck.

224 Old Brompton Rd. S. W. 5.

BIBLIOGRAPHY

Asherson, N.: J. Laryng. & Otol. 64:747 (Dec.) 1950.

Barré, J.: Rev. d'oto-neuro-oculist., Vol. 4, June, 1926.

Bedford, P. D.; Bosanquet, F. D., and Russel, W. R.: Lancet 2:55 (July 12) 1952.

Brain, W. R.; Northfield, D., and Wilkinson, M.: Brain 75:187 (June) 1952.

Epstein, J. A., and Davidoff, L. M.: Surg. Gynec. & Obst. 93:27 (July) 1951.

Exner, G.: Die Halswirbelsäule: Pathologie und Klinik, Stuttgart, Georg Thieme Verlag, 1954.

Falk, P.: Arch. Ohren-, Nasen- u. Kehlkopfh. 150: 1, 1941.

Ztschr. Hals- Nasen- u. Ohrenh. 46:257, 1939.

Goligher, J. C.: Lancet 1:985 (June 19) 1948.

Heck, C. V.: Surg. Gynec. & Obst. 102:657 (June) 1956.

Henschel, C.: Acta oto-rhino-laryng. belg. 6:341, 1952.

Hofer, G.: Monatsschr. Ohrenh., Vol. 58 (April) 1924.

Jackson, R.: The Cervical Syndrome, Springfield, Ill., Charles C Thomas, Publisher, 1956.

Laskiewicz, A.: Rev. laryng. 74:514 (Sept.-Oct.) 1953.

Lindemann, K., and Kuhlendahl, H.: Die Erkrankungen der Wirbelsäule, Stuttgart, Ferdinand Enke, 1953.

Maspetiol, R.; Chardin, and Millard: Ann. otolaryng. 71:451, 1954.

May, E.; Magder, and May, E. M.: Bull. et mém. Soc. méd. hôp. 55:1153 (July 17) 1939.

Passe, E. R. G.: Internionál Oto-Rhino-Laryngological Congress, London, 1949.

Acta oto-laryng. 42:133, 1952.

Piquet, J.: Ann. oto-laryng. 68:697, 1951.

Schindel, E.: Wien. med. Wchnschr. 101:235 (March 31) 1951.

Schlachter, A. A.: Nederl. tijdschr. geneesk. 77: 4683 (Oct. 14) 1933.

Schlomka, G., and Schröter, G.: Ztschr. ges. inn. Med. 8:473 (May 15) 1953.

Seiffert, A.: Ztschr. Hals- Nasen- u. Ohrenh. Vol. 27, Feb., 1930.

Spillaine, J. D., and Lloyd, G. H. T.: Brain 75: 177 (June) 1952.

Symonds, C.: Lancet 1:451 (March 7) 1953.

Tarneaud, J.: franç. Oto-rhin.-laryng. 1:173, 1952.

Laryngitis chronica, Paris, Masson & Cie, 1954.

Taylor, A. R.: Lancet 1:717 (April 11) 1953.

Vastine, J. H., and Vastine, M. H.: A. M. A. Arch. Otolaryng. 55:1 (Jan.) 1952.

Wiethe, C.: Ztschr. Laryng. Rhin. Otol. 24:54 (Jan.) 1933.

Acoustic Neurilemoma

KENNETH CAMBON, M.D., and FREDERICK R. GUILFORD, M.D., Galveston, Texas

History

The condition known as acoustic neurilemoma was first described by Sandefort of Leyden in 1777: he describes postmortem findings of what was probably an acoustic neurilemoma. Several other questionable reports of such lesions appeared in the next few decades. However, it is to that pioneer of neurology, Charles Bell, that we owe our first clinical-pathological presentation of what is without any question a case of acoustic neurilemoma. Together with the help of John Whiting in 1830 such a report was published as an appendix to his classical monograph on the nervous system. It remains such a masterpiece of clinical observation that little since has been added from the point of view of symptom description of the advanced case. Part of this is reproduced here in John Whiting's letter to Bell [5]:

> I found that she still had a distressing sensation on the left side of her face, etc., although altered in its character; her speech has become indistinct, her face was drawn to the right side, the masseter and temporal muscles of the left side had ceased to act, the tongue was protruded toward the left side, the hearing of the left ear had ceased; she could raise the left upper eyelid by voluntary power, but could not keep it elevated; the effort to raise the globe of the eye was attended with headache and giddiness; there was considerable secretion of tears; she was emaciated and bedridden, and complained of great and constant pain at the back of her head.
>
> About a month before her death her intellect became confused, her breathing difficult, her speech quite indistinct, and her deglutition impeded; she occasionally ground her teeth with violence, and her jaws were often firmly clenched, apparently by the contraction of the muscles of the right side; she seemed to die at length (in February, 1829) from difficult respiration, and want of the power of swallowing.

John Bright, whose name has been immortalized in renal pathology, some years later also described a similar case. In France, Cruveilhier devoted much time to this problem and since then there have been no major contributions to the description of the gross pathology.

It remained for Harvey Cushing to point the way to surgical diagnosis and treatment. He stressed that acoustic tumors, owing to the characteristic chronology of their symptoms, may as a rule be sharply distinguished from all other tumors of the cerebellopontine angle. He noted that tumors growing in the internal auditory meatus produce sensory disturbances first and motor manifestations later, whereas the reverse is true of the tumors growing toward the meatus. His book published in 1917, "Tumors of the Nervus Acusticus," is a model of scholarship.[5]

A review of the literature suggests that it is only in recent years that otology has made any contributions in this field. This would seem a sad reflection on otology, considering that almost invariably ear symptoms precede all others. Perhaps even more disturbing is the revelation by Lundborg (1950), who on reviewing 300 cases found that only 10% were diagnosed by otologists.[1]

An attempt will be made here to explain these diagnostic difficulties in terms of clinical findings and the pathological physiology of the lesion. Nevertheless, it is evident that there can be no substitute for a high index of suspicion, since these cases almost invariably present at first with symptoms caused by eighth nerve dysfunction. Since the surgical procedure is considerably less formidable if the lesion is discovered early, the otologist should play an important role in the diagnosis of this condition.

The common clinical findings in this condition have been succinctly charted in an

Submitted for publication June 7, 1957.

excellent article by Brown, Love, and Adams (1952).[2] Two of their tables listing first the symptoms caused by eighth nerve dysfunction and secondary other common neurological findings are presented.

Clinical Findings

(a) Symptoms Resulting from Dysfunction of the Eighth Nerve (Table 1).—Almost invariably such symptoms are the initial complaints of cases of acoustic neurilemoma. Hearing loss and tinnitus are much more predominant than vertigo, which may be entirely absent. Furthermore, in the early stage of the disease, the hearing loss may vary considerably from time to time and even show periods of returning to near normal levels. The nystagmus has been described by many authors with almost as many various "typical types of nystagmus" as writers. These symptoms can precede the development of symptoms suggesting intracranial disease by as much as by 22 years, the average being 1 to 3 years. Then, this disproportion between cochlear and vestibular symptoms, the waxing and waning of symptoms, and the uncertainty of the type of nystagmus may partially explain the poor diagnosis rate by otologists. Yet, as will be shown later, this apparent contradictory symptomatology becomes much more rational when interpreted on the basis of pathological changes.

(b) Findings in Addition to Dysfunction of the Eighth Nerve (Table 2).—It is immediately evident that corneal anesthesia is a common finding in this condition. Also worthy of note is the relatively low number with positive roentgenologic findings, only 42%. Denny has suggested that this could be improved with tomography of the internal auditory meatus.[3]

TABLE 1.—*Symptoms Resulting from Dysfunction of the Eighth Nerve in 150 Cases of Acoustic Neurofibroma* *

Symptom	Number	Per Cent (of 150)
Hearing loss or tinnitus as first symptom	105	70
Total hearing loss on side of lesion	85	57
Severe but not total hearing loss on side of lesion	29	19
Moderate hearing loss on side of lesion	19	13
Bilateral hearing loss	6	4
Islet hearing loss on side of lesion	1	1
Hearing loss beyond 4096 frequency	1	
Tinnitus	100	67
Vertigo or questionable vertigo	16	11
Dizziness	53	35

* Reprinted, with permission, from Brown, H. A.; Love, J. G., and Adams, N. D.: Otologic Evaluation of Unilateral Acoustic Neurofibroma, *Laryngoscope* 62:250, 1952, published by The Laryngoscope, St. Louis

TABLE 2.—*Findings in Addition to Dysfunction of the Eighth Nerve That Were Suggestive of Acoustic Neurofibroma in 150 Cases* *

Symptom or Sign	Number	Per Cent (of 150)
Corneal anesthesia	126	84
Ipsilateral	110	73
Bilateral	16	11
Facial paresthesias	77	51
Headache	105	70
Facial Weakness	55	37
Moderate	9	6
Complete	1	1
Spontaneous nystagmus	125	83
Choked disk	74	49
Facial pain	16	11
Visual disturbances	47	31
Positive roentgenologic findings	64	43
Positive Romberg sign	106	71

* Reprinted, with permission from Brown, H. A.; Love, J. G., and Adams, N. D.: Otologic Evaluation of Unilateral Acoustic Neurofibroma, 62:250, 1952, published by The Laryngoscope, St. Louis.

(c) Audiometic and Caloric Findings.—1. Caloric Testing: Almost invariably there is a nonfunctioning or hypoactive labyrinth on the ipsilateral side. There may also be hyper- or hypoactive changes on the other side. Again this apparent paradox has a sound pathological foundation. However, Dix and Hallpike report four cases with normal caloric responses[4] and Kristensen (Copenhagen) states that 10% of cases have a normal caloric response.[4]

Nevertheless it is clear that caloric testing must be one of the most useful tools of investigation available to the otologist. Too often this is not done until after more ominous cranial nerve palsies develop. It is our contention that in dealing with such lesions, one should use a sensitive, measurable, and repeatable test, such as the Cawthorne-Fitzgerald-Hallpike test. Not only does this test reveal early diminution in caloric response that grosser tests might not detect, but it will also demonstrate directional preponderance if present. This phenomenon may occur early in the course of acoustic neurilemoma[7] (and, of course other conditions as well), and cannot be discov-

ered when the conventional cold caloric test is used.

2. Audiometric Findings: As a general rule, these present as a pure nerve loss, but no typical audiometric pattern can be described. Indeed several cases of conductive type losses have been described with acoustic neurilemoma.[7] It is said that in the early stages low-tone losses are common.

The one distinct contribution of otology to this problem has been that of Dix, Hallpike, and Hood. They observed that recruitment of loudness was present in end-organ lesions, but was absent in tumors of the cerebellopontine angle. In general, this has proved to be true and is a valuable adjunct to the diagnosis. Other specialized tests such as those for diplacusis, threshold fatigue, aural overload,[8] and difference limen tests can help to confirm whether the hearing loss is caused by end-organ changes or not. Such investigation must be tempered with the realization that the tumor by affecting the vascular supply can eventually damage the cochlea and cause end-organ changes. In addition to this, it is possible for the patient with acoustic neurilemoma to suffer coincidentally from degenerative changes of the cochlea common to the older age group. It is suggested that those who condemn recruitment and other such tests on the basis of some apparently wrong predictions have done so prematurely.

(*d*) *Pathology.*—The etiology is unknown, but there would seem to be some hereditary factor. Gardner exhibited an elaborate family tree of five generations of more than 200 members, demonstrating transmission of these tumors as a true Mendelian dominant affecting 37 members of the family.[12] Trauma and pregnancy have also been suggested as inciting factors. The tumor is commoner in the female and appears in the prime of life, usually between 30 and 50. Cushing feels acoustic neurilemoma is much commoner than realized and that it is often missed at autopsy as the internal auditory meatus is so seldom examined.[5] This impression was confirmed by the findings of

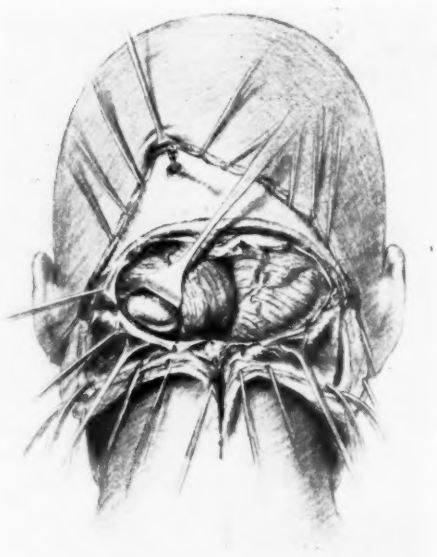


Fig. 1.—Showing exposure of encysted fluid within the arachnoid overlying a tumor, from a sketch during operation. (From Cushing, H.: Tumors of the Nervus Acusticus and the Syndrome of the Cerebellopontile Angle, W. B. Saunders Company, Philadelphia.)

Hardy and Crowe, who found six instances of acoustic neurilemoma in 250 consecutive routine autopsies.[10] None of these cases had any history of neurological disturbances. It is usually stated that it makes up from 6% to 8% of all intracranial neoplasms. Grossly the tumor can vary in size from a pinhead to an orange. It is either yellowish or gray in color. It is always encapsulated, and may be solid, cystic, or myxomatous. Almost invariably it is covered by an arachnoid cyst, which may reach surprising dimensions. This cyst is probably formed by the tumor impinging on the flocculus, thus impeding the proper evacuation of the lateral cistern. However, the tuft of choroid plexus in this region continues to pour fluid into the cistern so that a cyst is formed.

This arachnoid cyst can reach sizable dimensions (Fig. 1) and create a pressure effect of its own on the surrounding structures. Furthermore, these cysts may burst or evacuate spontaneously from time to time. Both the cyst and the tumor proper may

exert pressure on the internal auditory artery and cause labyrinthine symptoms.

The tumor originates in the internal auditory meatus, but grows out of it so that it is subdural, extracerebellar, and lies within the arachnoid capsule.

The tumor can distort the cerebral nerves and brain. The fifth nerve usually has an intracranial course of 1.5 cm. It may be stretched to over 5 cm. It is remarkable that the seventh nerve escapes so often as it does, since it has to encircle the upper pole of the tumor. Bell, in his original description in 1830, attributes this to its hardness in contrast to the soft texture of the nervus acusticus. This difference in texture can be readily appreciated in the autopsy room, and no better reason has been advocated. The sixth nerve almost never comes in contact with the growth, yet a unilateral abducens palsy is common. Cushing attributes this to the same type of vascular constriction to which has been attributed the internal squint so frequently observed in cerebellar tumors of other types.[5]

The 9th, 10th, and 11th cranial nerves are much nearer the facial and acoustic than is the trigeminal nerve, and also their foramen of exit is nearer the porus than is the trigeminal foramen. It would seem that they might be compromised at a comparatively early stage; yet such is not apparent clinically. Cushing feels that they probably are affected early, but that such functional impairment is difficult to detect in contrast to the obvious changes of a slight emotional asymmetry of the face or an easily appraised loss of corneal reflex.

Our own experience does not bear this out. The 9th nerve is difficult to test, but if one accepts laryngeal function and sternomastoid and trapezius movement as indices of the state of the 10th and 11th nerves, then involvement of these occurs infrequently and only late in the course of the disease. Dr. K. Earle of the Department of Pathology, University of Texas, has suggested that perhaps the strong pulsations of the vertebral arteries tend to encourage the tumor to take the line of least resistance forward toward the fifth nerve instead of backward.

Pontine and cerebellar compression also may occur. This can superimpose symptoms which can confuse the clinical picture.

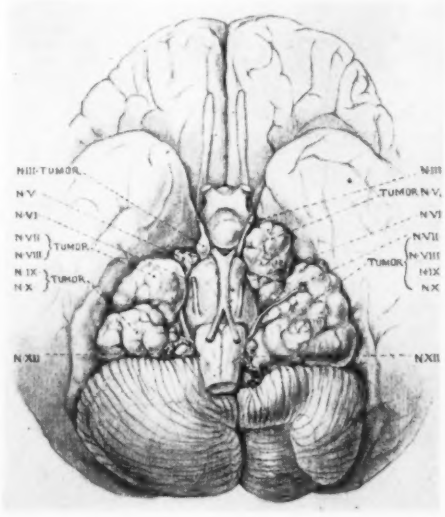
Obstructive dilatation of the ventricles may lead to more distant effects, resulting in papilledema and cerebral herniation, either through the foramen magnum or minor cortical protrusions which occur at the situations of the arachnoid villi.

Rarely multiple tumors may occur involving several cranial nerves (Fig. 2). These may be a manifestation of generalized neurofibromatosis. When this is associated with cutaneous pigmentation and the presence of multiple, sessile pedunculated tumors of the skin, the condition is known as von Recklinghausen's disease.

In summary, then, the gross intracranial changes which an acoustic tumor is capable of producing are of three kinds[5]:

1. Those relating to the common seat of origin of the growth in the meatus

Fig. 2.—An example of multiple and bilateral cerebral nerve tumors in a child of 11 years with inherited generalized neurofibromatosis. Bilateral deafness before death. Redrawn from Berggrün. (From Cushing, H.: Tumors of the Nervus Acusticus and the Syndrome of the Cerebellopontile Angle, W. B. Saunders Company, Philadelphia.)



2. Those relating to the nervous structures in the cerebellopontine angle
3. Those which are distant and are attributed to internal hydrocephalus.

Histology

There are more than 25 different names for this lesion, each given by such famous pathologists as Virchow and Rokitansky to bear out his particular view of the origin of the tissue. Indeed, in the excellent section on acoustic neurilemoma in "Tumors of the Peripheral Nervous System" by A. P. Stout [9] five lines are taken up to recite the different synonyms.

The issue is perhaps not settled yet. Some pathologists consider the tumor to arise from embryonic glial tissue which extends along the nerve from the brain stem. Since the vestibular portion of the eighth is the shortest sensory nerve in the body, there is more potential here than elsewhere. Because of the presence of collagen, Penfield believes these tumors should be called perineural fibroblastoma, as he maintains only fibroblasts can make collagen. Others consider it to arise from the sheath cells about the nerve; hence Fowler proposed the term neurilemoma, indicating sheath involvement and not fiber participation. This name has been accepted by the Armed Forces Institute of Pathology and is attaining a more universal usage. The histological picture is a combination of fibrous zones in which the nuclei tend to dispose themselves in palisades of whorls, together with a loose reticular tissue containing glia-like fibrils in which fat and hyaline metamorphosis commonly occurs. The solid part of the tumor is designated as Antoni Type A tissue; the loose meshwork is called Type B tissue. This division would appear to be an artificial one, as tissue culture work has shown that the cells from the two areas are all Schwannian.[9] Occasionally the tumor may be rather vascular, a fact of some clinical as well as surgical significance (Fig. 3).

Histological examination of the labyrinth shows no change other than some extravasation and pigmentation due to stasis. The organ of Corti and vestibular organs do not degenerate. The vestibular nerve degenerates only as far as the vestibular ganglion. This is not true when the tumor compromises the blood supply of the labyrinth; then, of course, labyrinthine degenerative changes occur. On occasion the tumor itself has invaded the labyrinth proper, but this must be a rare occurrence.[11]

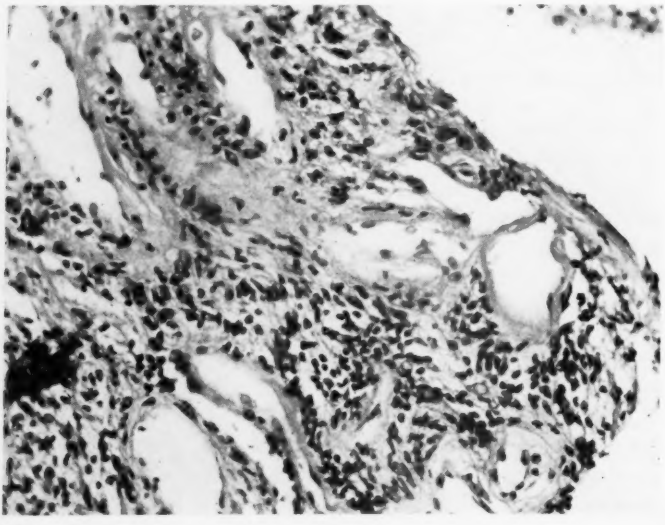


Fig. 3. — Photomicrograph of section of acoustic neurilemoma.

Clinical-Pathological Correlation

It may at first seem puzzling that so few patients initially complain of vertigo. The apparent reason for this is that the tumor destroys the nerve function slowly enough to permit compensation by the contralateral labyrinth. Hence deafness and tinnitus are usually the presenting symptoms.

The traditionally described low-tone hearing loss has been explained on the basis of the peculiar arrangement of nerve fibers in the vestibular portion of the eighth nerve. The fibers carrying the low tones supposedly are the central core of the nerve; those carrying the higher tones are arranged in a spiral fashion around the nerve and thus are more flexible and more immune to pressure effects. (Our own modest study had only one case that presented as a low-tone loss. See audiogram of Case 3, Fig. 5.) It is possible that some of the conductive losses described in the literature may have been coincidental and not caused by the neurilemoma.

The periodic exacerbations and remissions of symptoms are caused by the changes in size of the arachnoid cyst surrounding the tumor. Presumably the pressure changes could also produce spasms of the labyrinthine vessels and thus mimic hydrops of the labyrinth. This also explains why lumbar puncture may give profound relief, dangerous as this procedure is.

The spontaneous gaze nystagmus, its variability, and the fact that it is seldom of a true labyrinthine type can be explained as follows: At the very initial stage there is a true irritation of the vestibular nerve; in time with progression of the tumor, degeneration occurs. Later the tumor and the unstable arachnoid cyst about it press against the vestibular nucleus on the brain stem. As the tumor grows it exerts pressure on the cerebellum, which superimposes the cerebellar form of nystagmus on its predecessor. Finally an increase in cerebrospinal fluid pressure may produce changes in the contralateral labyrinth. Thus one can in truth expect any form of spontaneous nystagmus with this lesion.

It is of interest to note that at the University of Texas Hospital since 1900, of more than 500 intracranial neoplasms that came to Pathology, 25 were diagnosed as acoustic neurilemoma. Most of these seem to have occurred in fairly recent years, perhaps showing that there is an increased interest in the subject. A review of the charts demonstrated that in the earlier years often the patients were not referred to an otologist, as the patients presented as full-blown, far-advanced cases. In general, our cases fitted well into the pattern of symptomatology as described by Brown, Love, and Adams[2] (Tables 1 and 2). Two of these had normal responses to the mass caloric test, an incidence of 8%. There were two surgically proved cases of bilateral involvement, and one apparent clinical case in which the other side has yet to be explored. Five of the cases showed normal protein levels in spinal fluid examination. Four of the more unusual cases are presented here, as well as a recent case that was referred to our department with a tentative diagnosis of acoustic neurilemoma, which investigation proved to be wrong.

This first case is described because of the rather unusual presenting symptoms:

CASE 1.—A 51-year-old white woman was first seen in the OPD on Feb. 7, 1947, with a primary complaint of "weakness in the right hip, which caused her to fall to the right." She was extensively investigated in the Orthopedic Department, and, since no bony pathology was found, she was referred to Medicine Clinic. Here she was labeled as "psychoneurosis-hysteria" and referred to Neurology Clinic, where it was noticed she had papilledema. Further examination now revealed she had a left facial weakness and an absent corneal reflex on the left. There was no record of a loss of hearing, but neither audiogram nor caloric tests were done.

Skull x-rays were normal.

Spinal fluid showed 100 mg. protein per 100 cc.

Craniectomy revealed an acoustic neurilemoma.

The following case is described to illustrate the importance of early caloric testing:

CASE 2.—This 52-year-old white woman was first seen in 1952. She complained of tinnitus of the left ear and a marked loss of hearing in the

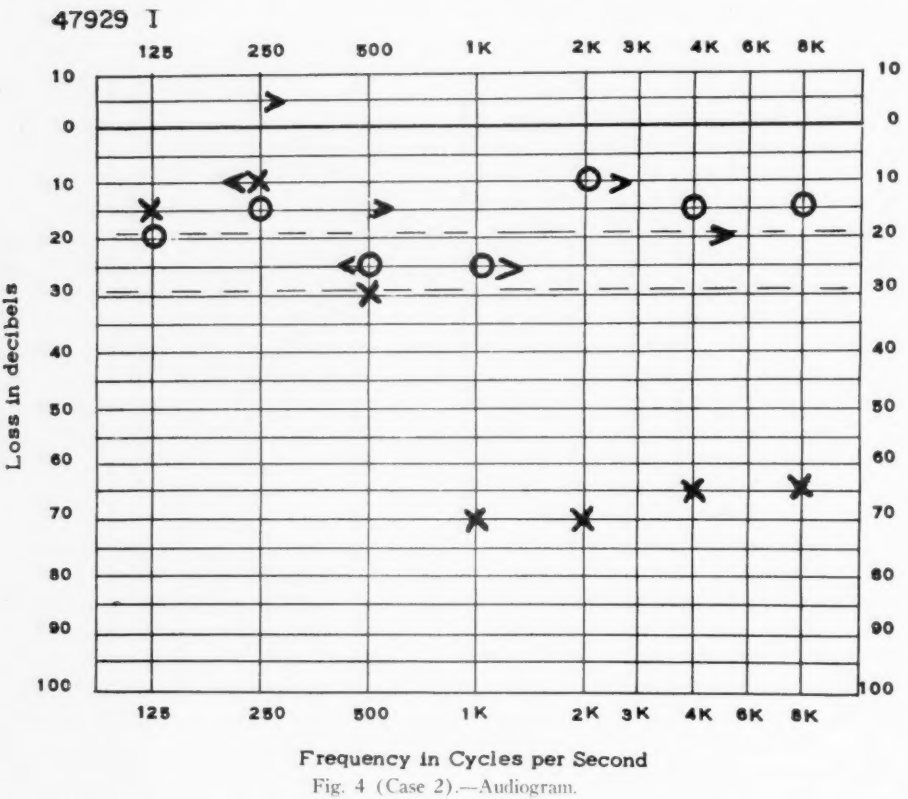


Fig. 4 (Case 2).—Audiogram.

same ear. She also had occasional bouts of vertigo. At age 16 she had had a severe bout of otitis media with a central perforation of the right tympanic membrane. Otherwise her history was negligible.

Physical Examination.—ENT: Clinical examination revealed a healed central perforation of the right tympanic membrane. Otherwise ENT examination was normal.

Audiogram: Showed a 57 db. loss in the left ear mostly in frequencies 1k, 2k, 4k, and 8k (Fig. 4). Recruitment tests were not done.

X-rays: Showed no abnormalities of the internal auditory meatus.

Caloric tests: Not done at this time.

A tentative diagnosis of thrombosis of some labyrinthine vessel was made. The patient was treated symptomatically with vitamin B_{12} and vasodilators, with temporary relief.

She was referred to a neurologist, who concurred in the diagnosis.

On return of the symptoms she went to a large Eastern clinic, where a similar diagnosis was made and the same treatment instituted with substantially the same results.

In December, 1956, more than four years later, she returned with the same complaints but now also stated she had a loss of sensation on the left side of her face and loss of taste on the left side of her tongue.

It was now noted she had no corneal reflex on the left side.

Caloric tests were done. The right side gave a normal response, whereas the left side showed no response to the mass test.

Repeat x-rays (Stenver's views) now revealed enlargement of the right internal auditory meatus.

Spinal fluid studies showed no abnormalities.

The patient underwent surgery, at which time a large acoustic neurilemoma was removed.

This would seem to lend emphasis to the suggestion of Denny: "Repeated examination is often essential before a correct diagnosis can be made, and patients with unilateral unexplained deafness, associated with labyrinthine hypofunction, should be subjected to repeat examinations." [3]

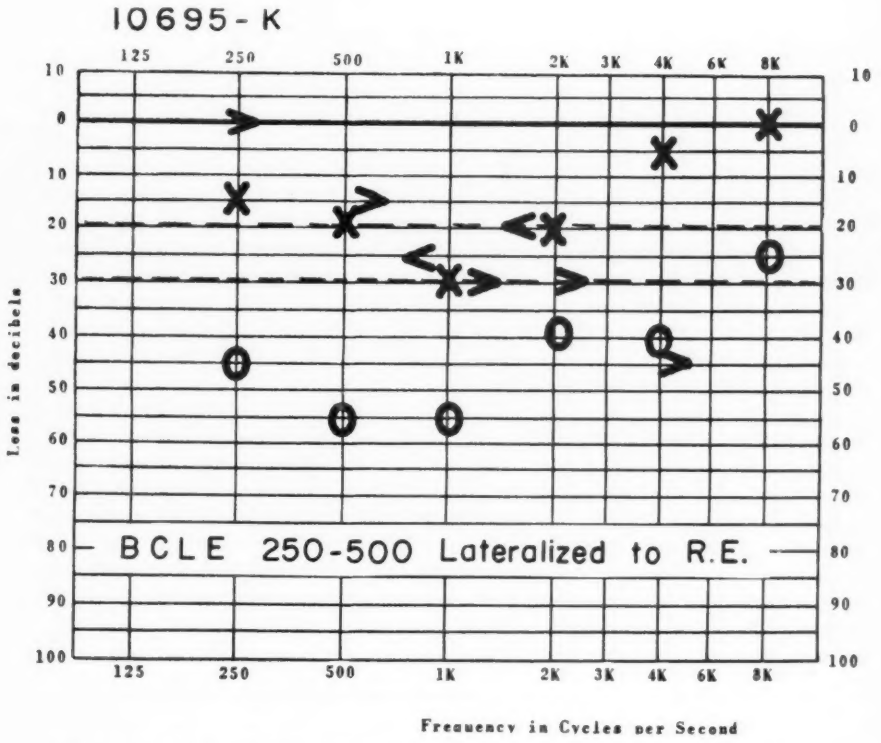


Fig. 5 (Case 3).—Audiogram illustrating a conductive hearing loss in a case of acoustic neurilemoma.

The following case is presented to illustrate a case of acoustic neurilemoma with a conductive loss, with no other ear pathology evident:

CASE 3.—A 54-year-old white woman was admitted July 7, 1954, with a history of severe tinnitus, severest on the left, bilateral loss of hearing, dizzy spells, difficulty in speech, dysphagia, headaches, and vomiting, all of about three months' duration.

Physical Examination.—ENT: Reported as normal. (Apparently the larynx was not examined.)

Eyes: Horizontal nystagmus, diplopia on left lateral gaze, left upper lid lag, loss of corneal reflex on the left.

Neurological: Loss of sensation in the area of distribution of the left maxillary nerve.

Skull x-rays: Normal

Audiogram: Reported on a 50 db. loss on left side with a large conductive element; 23 db. loss on right side (Fig. 5).

Caloric tests: Normal response bilaterally to mess test.

Spinal fluid: Protein 198 mg. per 100 cc.

Craniectomy was done and a neurilemoma was found and reported as nearly completely removed.

The following case is presented to illustrate how coincidental end-organ degenerative changes can occur:

CASE 4.—A 46-year-old white man was seen in consultation in 1955. He complained of tinnitus and deafness of the right ear of seven to eight years' duration. In the past six months he had noticed some slight numbness of the right side of his face and a thick feeling in his tongue on that side. At the same time he noticed some tinnitus and loss of hearing in the left ear. He complained of some difficulty in swallowing.

The patient denied having any diplopia but did complain of periodic severe headaches.

Physical examination revealed no ENT abnormalities.

Corneal reflex was absent on the right side, as was sensation to all areas supplied by the three divisions of the trigeminal. There was also marked right masseter weakness.

Fundoscopic examination was normal; however, the left pupil was 2 mm. larger than the right.

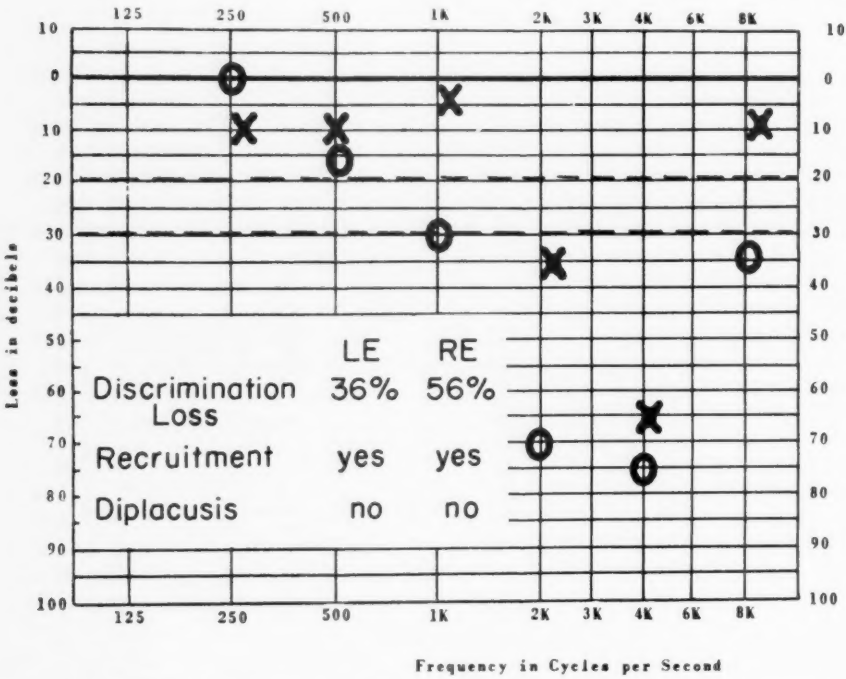


Fig. 6 (Case 4).—Audiogram to illustrate presence of recruitment in a case of acoustic neurilemoma.

There was a coarse nystagmus present on lateral gaze to either side.

Caloric tests were done. There was no response to the mass test on the right side and a depressed response on the left.

An audiogram done at this time showed a 17 db. loss on the left and a 38 db. loss on the right. Both ears had poor discrimination, 64% and 44% respectively. Both showed recruitment (Fig. 6).

Routine x-rays of the skull were normal, but Stenver's projections showed apparent enlargement of the right internal auditory meatus.

A ventriculogram was reported as follows: ". . . there is a dilatation of the lateral and third ventricles without displacement, with evidence of compression of the right portion of the fourth ventricle and aqueduct, probably due to a right cerebello-positive angle lesion."

Spinal fluid studies were normal—the protein level being only 10 mg. per 100 cc.

EEG was a normal tracing.

Surgery revealed an acoustic neurilemoma on the right side somewhat larger than a walnut, with several overlying cysts.

The following case is presented to demonstrate the aphorism that the patient must be reviewed as a whole:

CASE 5.—A 45-year-old white man was referred for consultation to consider the possibility of a cerebello pontine angle lesion or a nasopharyngeal lesion. He complained of chest pain, tinnitus in the left ear, with some loss of hearing and vertigo, all of six months' duration. He also complained of increasing hoarseness. He had lost 25 lb. in the past four months.

ENT examination was negative except for a left recurrent laryngeal paralysis. Corneal reflexes were normal. Tuning fork tests showed a left nerve loss for the higher frequencies that was confirmed by audiogram (Fig. 7A).

Cawthorne-Fitzgerald-Hallpike caloric tests revealed a moderate left canal paresis (Fig. 7B).

Chest x-ray had been reported as normal.

Spinal fluid studies showed an elevated protein of 102 mg. per 100 cc., with normal pressure.

This case, then, presented with many of the diagnostic criteria of acoustic neurilemoma.

However, in view of his history of chest pains, weight loss, and the fact he was a heavy smoker, a repeat chest x-ray was done. This revealed ". . . a rather faint circumscribed infiltration in the left upper lobe."

Bronchoscopy was performed by Dr. G. McReynolds, who did a biopsy of a small edematous

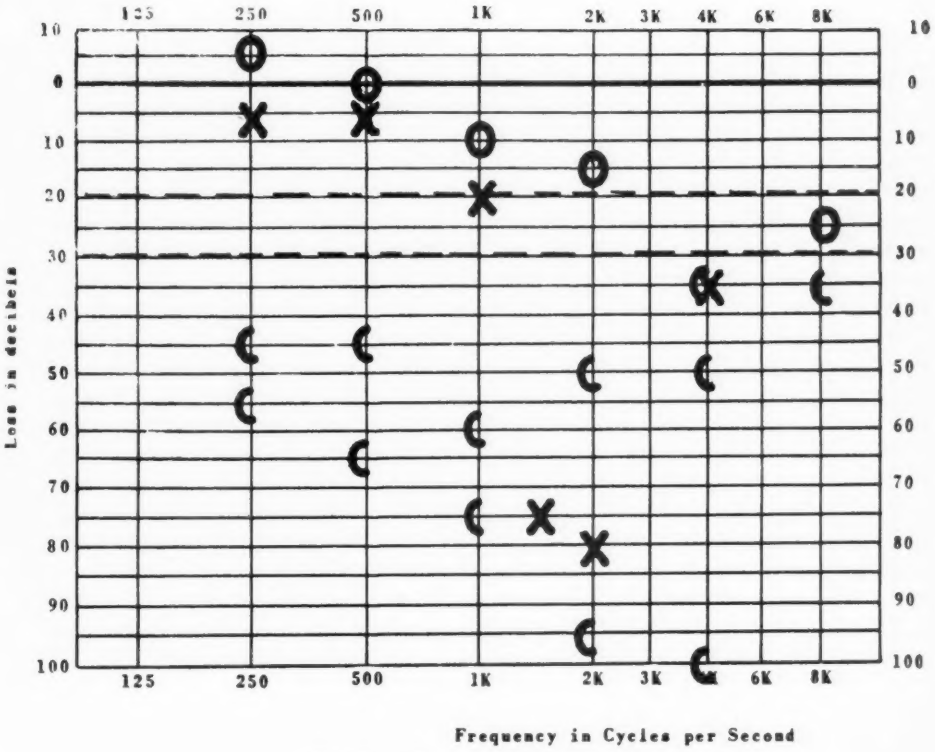


Fig. 7A (Case 5).—Audiogram.

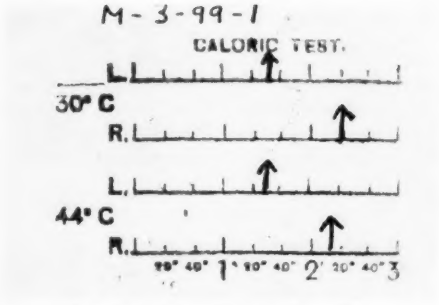


Fig. 7B (Case 5).—Calorogram.

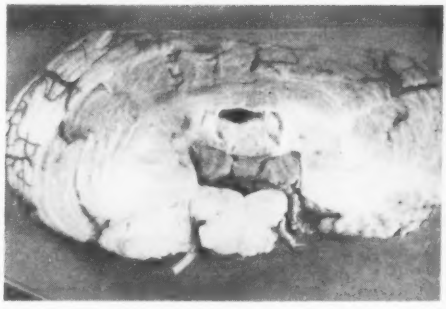


Fig. 8 (Case 5).—Illustrating involvement of left cerebrellopontine angle with a metastatic tumor mass.

area on the interior part of the proximal spur of the left upper lobe bronchus. The report came back as squamous-cell carcinoma.

The patient died four weeks later. Autopsy revealed cerebral metastases. One area involved the left cerebellopontine angle (Fig. 8).*

In the cases reviewed, diagnosis was difficult in the early stages, and the true nature of the lesion was often mistaken for more mundane conditions such as sinusitis, anxiety states, and Ménière's syndrome. Few had the benefit of audiometric and caloric tests until at least two years after the initial symptoms.

This would seem to emphasize the point made in almost every article of modern

* Courtesy of Dr. K. M. Earle, Department of Pathology, University of Texas.

vintage, that only with the more widespread intelligent application of modern audiometry and caloric testing can acoustic neurilemoma be suspected at the desirable early stage. To this might be added the aphorism that in cases of unexplained hearing loss, tinnitus, and/or vertigo no otological examination is complete without an assessment of the other cranial nerves.

University of Texas Medical Branch.

REFERENCES

1. Lundborg, T.: Nord Med. 44:1520, 1950.

2. Brown, H. A.; Love, J. G., and Adams, N. D.: Laryngoscope 62:250, 1952.

3. Denny, W. R.: J. Laryng. & Otol. 69:608, 1955.

4. Dix, M. R.; Hallpike, C. S., and Hood, J. D.: Proc. Roy. Soc. Med. 42:527-530, 1949.

5. Cushing, H.: Tumors of the Nervus Acusticus and the Syndrome of the Cerebellopontile Angle, Philadelphia, W. B. Saunders Company, 1917.

6. Kristensen, H. K.: Acta Psychiat. et neurol. scandinav. 27:287, 1952.

7. Dix, M. R., and Hallpike, C. S.: Proc. Roy. Soc. Med. 43:291, 1950.

8. Yantis, P. A.: J. Speech & Hearing Dis. 21:303, 1956.

9. Stout, A. P.: Tumors of the Peripheral Nervous System, Atlas of Tumor Pathology, Sec. 2, Fasc. 6, Armed Forces Institute of Pathology, 1949.

10. Hardy, M., and Crowe, S. J.: Arch. Surg. 32:292, 1936.

11. Scott, P.: J. Laryng. & Otol. 53:772, 1938.

12. Eggston, A. A., and Wolff, D.: Histopathology of the Ear, Nose and Throat, Baltimore, Williams & Wilkins Company, 1947.

Mixed Tumors of the Palate

FERNAND MONTREUIL, M.D., Montreal

James Paget was the first to draw attention to mixed tumors outside of the major salivary glands, in 1851. Since this time, mixed tumors have been reported to arise from numerous areas: eyebrow, cheek, pharyngeal wall, tongue, and even in distant areas such as the leg and the hand, but these are most exceptional sites of origin.

Outside the major salivary glands, they are, by far, commoner in the upper respiratory tree; Hayes Martin has stated that "the palate is the site of origin of a greater percentage of benign mixed tumors of the salivary gland type, than any other structure of the head and neck, except the parotid salivary gland." Cordray reported that in 61 cases of mixed tumors seen during a period of 10 years, at the tumor clinic of the Massachusetts Eye and Ear Infirmary, 7 were in the palate. Owens, reviewing the literature over a period of 20 years, found 1138 such tumors outside the major salivary glands, of which 264, or 23.3%, were in the palate. On the other hand, Howarth, in 1936, stated that mixed tumors constituted by far the commonest tumor that occurred in the substance of the soft palate.

In spite of the numerous case reports which have appeared in the literature through the years, these tumors are still often unrecognized and consequently mismanaged. It is for this reason that the following cases are reported.

CASE 1.—The first case is that of a 44-year-old man who consulted because of a mass in the left side of the soft palate (Fig. 1). The patient had become aware of its presence eight years before, when he was told of its existence, by his physician, during the course of a routine examination. The patient had never had any symptoms referable to this mass which, according to him, had not changed during this eight-year period. The left side of the palate was seen to bulge forward and, on palpation, a firm lobulated mass could be felt in the supra tonsillar region. The mass extended upward, behind the hard palate. The mucous membrane overlying this mass was normal in appearance. The tumor was felt to be immediately underneath the mucous membrane and was mobile. It might be emphasized at this point that no examination of the oral cavity is complete without a digital examination. The examining finger can, better than any instrument, appreciate the nature of a lesion, its consistency, its extension, its infiltration,

Submitted for publication July 17, 1957.

From the Ear, Nose and Throat Service, Notre Dame and Queen Mary Veterans' Hospitals.

Presented at the Sixth International Congress of Otolaryngology, Washington, D. C., May 7, 1957.

Fig. 1 (Case 1).

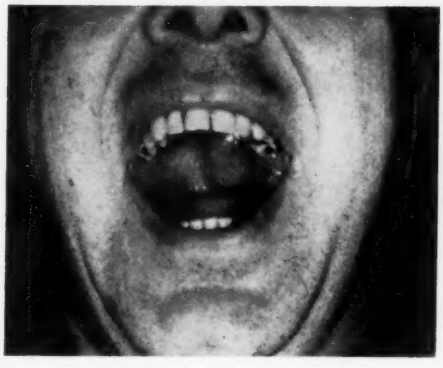


Fig. 2 (Case 2).—Surgical specimen.

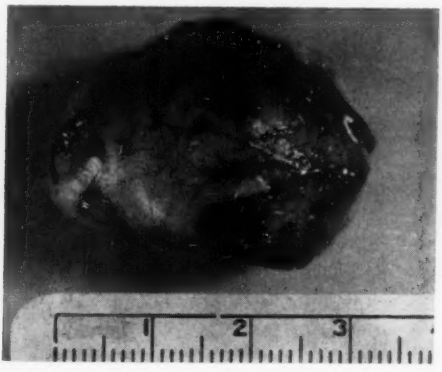


Fig. 3 (Case 2).

and finally its mobility. Excision of the tumor was performed via an oral approach, under general anesthesia, and a mass 3.5×2 cm. was removed with an intact capsule (Fig. 2). The sutures were removed on the fourth postoperative day, and the patient was allowed to go home.

CASE 2.—The second case is that of a 29-year-old man who had been aware of the presence of a mass behind his soft palate, for about three years. A course of radiotherapy had been given at some other hospital without any appreciable change in the tumor. The patient had no other complaint than fullness on the left side of the soft palate. Examination showed the presence of a deeply situated mass which made all of the left side of the soft palate bulge forward (Fig. 3). Palpation revealed a firm, lobulated mobile tumor under the substance of the soft palate. Excision, via an oral approach, was performed under general anesthesia, and a large 5.5×4×2.5 cm. tumor, which extended to the base of the skull, was removed with an intact capsule (Fig. 4). In this case, the large operative defect was loosely packed with gauze. The packing was removed the day following surgery, and the patient was allowed to leave the hospital on the third postoperative day.

The tissues of the mouth have remarkable healing properties; it is amazing to see how rapidly such a large defect will granulate in, and healing is usually complete within a week to 10 days.

CASE 3.—The third case, a 26-year old man, was referred to us after tonsillectomy and biopsy which reported a mixed tumor. The patient had consulted his physician because of a sudden increase in the size of a mass, first noted in his soft palate, some three months before. The sudden increase in size of the tumor, over a period of four to five days, had caused a feeling of fullness in the throat and some difficulty in swallowing. There had never been any pain. The patient was admitted to hospital, where a tonsillectomy was performed. At the time of the operation, it became obvious that the bulging of the palate was not caused by the tonsil, but was due to a mass behind the soft palate (Fig. 5). A biopsy specimen taken, in the supratonsillar region, was reported to be a mixed tumor. The patient was seen three days after tonsillectomy, and examination revealed a normally healing tonsillar fossa at the time. Above the tonsillar fossa all the soft palate was seen to bulge forward. A small area of the mucous membrane showed a superficial and irregular ulceration, with presence of granulation-like tissue in the middle of the ulceration; this represented the area where the biopsy specimen had been taken. Palpation, somewhat difficult because of pain, revealed a firm, lobulated mass occupying all of the left side of the soft palate and extending upward behind the hard palate. Excision of the tumor was performed three weeks later. At the

Fig. 4 (Case 2).—Surgical specimen.

Fig. 5 (Case 3).—Ulcerating surface can be seen in the supratonsillar area where biopsy specimen was taken.

Fig. 6 (Case 3).—Surgical specimen showing large area of palate mucosa.

time of the operation, care was taken to circumscribe and include the ulcerated area with the specimen. A fairly large portion of the mucous membrane of the soft palate was sacrificed along with the mass (Fig. 6). The tumor weighed 50 gm. and measured 6×4.5×3 cm. and was covered with an intact capsule, except for the area of the biopsy. The patient was discharged from the hospital on the third postoperative day with a normally healing wound.

Two other cases, occurring in a woman 45 years of age and in another woman of 39 years, were treated in the same fashion.

Dubecq and Maronneaud have expressed the opinion that mixed tumors of the palate occur more frequently in female patients. A review of available case reports does not suggest any sex preponderance, and in the present series two were female and three were male patients. These tumors occur more frequently between the ages of 20 and 40. It is usual to find that the tumor has been present for some time and has attained considerable size by the time the patient seeks treatment. The left side of the palate has been reported to be more frequently involved than the right side, and in this series the tumor was found to be on the left side of the palate in all five cases.

The lack of symptoms is well illustrated in the cases just summarized. The presence of a slowly growing, nontender mass in the palate is what brings the patient to seek advice. The growth, increasing in size in such an insidious manner, may and usually does attain considerable proportions by the time the patient seeks relief; it is not uncommon to have the tumor mass noted as an incidental finding during the course of a routine general or dental check-up. Pain is absent, and the tumor may remain dormant for years. There may occur periods of accelerated activity which will manifest themselves by a sudden increase in the size of the tumor. One must always seriously consider malignant degeneration in cases where a tumor, present for a long time, suddenly shows an increase in size, particularly when this is accompanied by pain and ulceration. As the tumor increases in size, the patient may experience some difficulty in wearing a previously well-fitting dental prosthesis; salivation may increase; a feeling of fullness in the mouth may appear; talking and swallowing may become interfered with. Mixed tumors, particularly of the hard palate, exceptionally wear through bone and invade the nasal fossa or the sinus. Pressure signs are otherwise very rare, and it is surprising that symptoms due to pressure on the Eustachian tube or on the cranial nerves, as the tumor extends to the base of the skull, do not make their appearance. On examination one might easily mistake a mixed tumor of the soft palate for quinsy. The tumor is never in midline. One side of the soft palate bulges forward and medially; the tonsil is pushed forward and downward; the uvula is shifted to the uninvolved side. Signs of inflammation are lacking, and the mucous membrane is usually normal in appearance, although one may at times see excessive vascularization of the mucous membrane overlying the tumor mass. Palpation with the finger reveals the firm consistency of the tumor, which often presents soft and even fluctuant areas. The finger will also determine the exact size and extension of the tumor mass and its mobility.

It is beyond the scope of this paper to enter into the long-discussion pathogenesis of mixed tumors. The French school of thought advanced the theory that these tu-

mors were of epithelial origin, and it is the theory that is generally accepted today.

Mixed tumors are characterized by the presence of epithelial and mesenchymal tissues. The preponderance of one or the other varies greatly. Epithelial cells, in strand or glandular formation, are intermingled in the midst of mesenchymal tissues of myxoid, fibrous, cartilaginous, or osseous characteristics. Epithelial cells present various degrees of differentiation before taking up the form and also the characteristic of mesenchymal cells (Fig. 7). One such cell is seen in the microphotograph. The cell is detached from the epithelial strand and has taken on a somewhat stellate form. Pathologists insist upon this cellular differentiation to make a diagnosis of mixed tumor. Many authors are of the opinion that mixed tumors arising outside the major salivary glands are more likely to undergo malignant degeneration than others.

A presumptive diagnosis of mixed tumor of the palate may be made from the history of a slowly growing, well-circumscribed, nontender, firm mass in which one may at times feel soft and fluctuant areas. The mass is felt immediately underneath the mucosa and is mobile. The final diagnosis, however, is made only after histological study. Biopsy is not recommended unless the tumor is ulcerated. We are in the presence of a clinically benign, well-circumscribed lesion, and it is of utmost importance to respect its capsule, so that the removal of the tumor can be made with an intact capsule. Needle biopsy is recommended by some authors. Had it been done in the case that follows, we should have known the true nature of the lesion we were dealing with and we should have been prepared to meet any eventuality which might have arisen during the course of surgery.

Case 4.—The patient was a 53-year-old man who complained of difficulty in swallowing because of a mass situated on the right of the soft palate (Fig. 8). The mass, present for some years, had slowly increased in size during the course of the last eight months. Examination and palpation suggested a mixed tumor, and its removal was recommended. The tumor was excised through the usual oral approach with the patient under general anesthesia. The mass was a large one, and dissection offered no difficulty until we reached the inferior and lateral pole. At this point, it was impossible to pass the finger or even an instrument between the tumor mass and the angle of the jaw. A lower molar tooth projected medially and rendered manipulations, along the inner surface of the mandible, most difficult. It was impossible to free the tumor at this point, and, as we feared to rupture the capsule by using too much traction, a tonsil snare wire was passed around the mass, which was then pulled free of its attachments. A most profuse hemorrhage ensued. The cavity was packed and the specimen examined. The hemorrhage was readily explained by the presence of an artery, 3 mm. in diameter and 1 cm. in length (Fig. 9). The vessel had been torn some distance from the tumor bed; it was consequently impossible to locate and clamp it. Ligation of the external carotid artery was necessary to control the hemor-

Fig. 7.—Photomicrograph reduced about 40% from mag. ×80. Cells detached from epithelial clumps have already taken on mesenchymal characteristics.

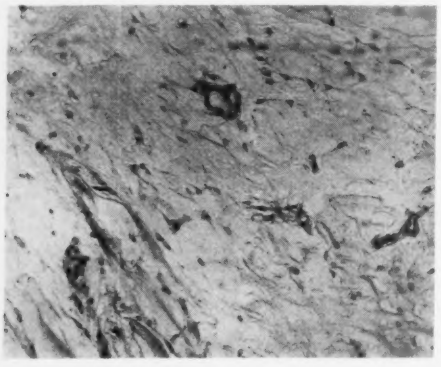


Fig. 8 (Case 4).

Fig. 9 (Case 4).—Surgical specimen. The artery may be recognized under tumor mass.

rhage. The tumor measured 6×4 cm. and had an intact capsule. Section revealed it to be a cavernous hemangioma (Fig. 10). In spite of considerable edema and hematoma of the soft palate and submaxillary space, which was such that a tracheotomy was seriously considered but not performed at that time, the patient made an uneventful recovery.

Mixed tumors are not radiosensitive, and, although cures have been reported following treatment with x-ray, radium, etc., it is generally accepted that surgical excision is the treatment of choice. We have found that the Davis mouth gag, with the patient under general anesthesia, offered a most adequate exposure. An incision of the mucous membrane over the most prominent portion of the tumor is made in an anteroposterior direction. The incision should be long enough in order to get adequate exposure of the tumor mass, which is often much larger than expected. The capsule is identified, and dissection is carried out with sharp or blunt instruments, the finger being by far the best. The mass is easily freed as there is a good line of cleavage; the tumor is removed with a minimal amount of bleeding, which is controlled by a few minutes of pressure.

Fig. 10 (Case 4).—Cut section of surgical specimen.

Preliminary ligation of the external carotid artery has been recommended by certain authors, but we think that this is not necessary. Excision of the tumor, via an external approach, has also been advocated but, unless the tumor be of such size as to make its removal via the mouth impossible, the oral approach is, by far, a simpler and more direct route. As mentioned previously, the postoperative course is most benign, with a minimal amount of discomfort and complete healing within a week to 10 days.

1123 St. Joseph Blvd., E.

BIBLIOGRAPHY

Anderson, W. A. D.: Pathology, Ed. 2, St. Louis, The C. V. Mosby Company, 1953, pp. 705-706.

Bompoint, J., and Rabechault, R.: Tumeur mixte de la voûte palatine, Rev. Laryng. 73:279, 1952.

Calmet, L., and Fabre, L.: Tumeur mixte du lobule du nez, Ann. oto-laryng. 67:500, 1950.

Campus, G.: Tumori misti del velopendolo derivati dal prolungamento faringeo della parotide, Minerva otorinolaryng. 5:84, 1955.

Chamberlin, W. B.: Mixed Tumors of the Salivary Gland Type Occurring in the Soft Palate, Tr. Am. Laryng. Rhin. & Otol. Soc. 33:175, 1927.

Chatellier, H. P.; André, P., and Neveu: Trois cas de tumeurs pharyngées (dont deux cas de tumeurs mixtes), Ann. oto-laryng. 69:362, 1952.

Cody, C. C., III: Venous Extension of Mixed Salivary Gland Tumor, Ann. Otol. Rhin. & Laryng. 61:384, 1952.

Cordray, D. P.: Mixed Tumor of the Region of the Head, Arch. Otolaryng. 49:631, 1949.

Cutler, M.: Mixed Tumor of the Palate: Late Results in 5 Cases Treated by Telecurie-therapy, Am. J. Roentgenol. 61:82, 1949.

Dubecq, X. J., and Maronneau, D. P.: Les tumeurs palatines d'origine glandulaire, Rev. stomatol. 47:345, 1946.

Ehrlich, H. E.: Mixed Tumors of Pterygomaxillary Space, Oral Surg. 3:1366, 1950.

Fry, R. M.: Structure and Origin of 'Mixed' Tumours of the Salivary Glands, Brit. J. Surg. 15:291, 1927.

Hendrick, J. W.; Ward, G. E., and Lacy, M. M.: Salivary Tissue Neoplasms, Am. J. Surg. 81: 373, 1951.

Howarth, W.: Tumours of the Palate, J. Laryng. & Otol. 45:673, 1930.

Some Tumours and Ulcers of the Palate and Fauces, Lancet 2:1139, 1936.

Joske, E. A.: A Mixed Tumour of the Palate with Sarcomatous Changes, M. J. Australia 2:347, 1946.

Koch, F.: Zum Vorkommen der Mischgeschwulste am weichen Gaumen, Wien. klin. Wchnschr. 40:780, 1927.

Lambert, V.: Mixed Salivary Tumours of the Upper Respiratory Tract, Proc. Roy. Soc. Med. 45:826, 1951-1952.

Layera, J., and Orosco, G.: Tumor mixto de velo de paladar, Prensa med. argent. 15:890, 1928.

Lederer, F. L.: Mixed Tumors of the Palate, in Diseases of the Ear, Nose, and Throat, Ed. 6, Philadelphia, F. A. Davis Company, 1952, pp. 1295-1296.

Lederman, M.: Mucous and Salivary Gland Tumours, Brit. J. Radiol. 14:329, 1941.

Martin, H.: Tumors of the Palate (Benign and Malignant), Arch. Surg. 44:599, 1942.

Mumma, C. S.: Treatment of Mixed Tumor of Tongue with Radioactive Chromic Phosphate Solution, Arch. Otolaryng. 58:192, 1953.

Owens, H.: Minor Salivary Gland Tumors in the Respiratory Tract and Ear, Laryngoscope 59: 960, 1949.

Pizzetti, F., and Leonardelli, G. B.: Sui tumori misti dell'estremo cefalico, Tumori 24:136, 1950.

Puente Duany, P.: Algunas notas sobre los mal llamados tumores mixtos del velo del paladar, Bol. Liga contra cáncer, 5:353, 1930.

Simard, L. C.: Tumour of the Palm Having the Structure of a Mixed Tumour of the Salivary Glands, Am. J. Cancer, 33:182, 1938.

Wilkerson, W. W., Jr.: Mixed Tumors of the Soft Palate and Parotid Gland, Laryngoscope 63:972, 1953.

Diagnosis and Management of Parotid Tumors

SAMUEL L. PERZIK, M.D., Beverly Hills, Calif.

It is commonly accepted that the management of parotid tumors calls for complete resection of the growth. But beyond this there is little agreement and the literature on the subject contains many contradictory statements. Perhaps the most vexing problem is whether mixed and low-grade mucoepidermoid tumors are benign or malignant. The decision is largely based on the likelihood of recurrence at the primary site; yet such recurrences depend not only on the growth potential of the tumor, but also on the surgical technique employed in extirpation of the mass. If any vestige of the diseased tissue is permitted to remain, the second appearance may clinically be classified as a recurrence, but, strictly speaking, it is a continuation of the uncontrolled original process.

The first prerequisite for successful management of parotid tumors is correct assessment of the extent of involvement. Often it is assumed that mere enucleation of "encapsulated" mixed tumors will suffice, but in an earlier paper I have presented "the case against enucleation" [1] and shown that such a procedure is inadequate, as scattered tumor cells may inadvertently be left behind. The hazard of injury to the facial nerve is no excuse for sacrificing the effectiveness of the operation. Through exposure of the facial nerve, facilitated by preoperative sialangiography, it becomes possible to extend the field of operation as far as required, without routinely going to the extreme of total parotidectomy. Other questions which have not yet been definitely settled are elective neck dissection at time of excision of the primary growth, and the place of radiation in the management of parotid tumors.

Submitted for publication Aug. 1, 1957.

Department of Surgery, Cedars of Lebanon Hospital, Los Angeles.

Read at the Sixth International Congress of Otolaryngology, Washington, D. C., May 7, 1957.

Method and Material

The understanding of parotid tumors and their management has been hampered by an unfortunate research set-up. Usually the solution of such problems is expected from a review of cases referred by a large number of clinicians, operated on by various surgeons, histologically studied by different pathologists, with final evaluation by one or several members of a writing team. In their analysis they are forced to interpret the findings of others as best they can, making due allowance for omissions in notations, and by grouping together findings of a variety of observers who may have used inconsistent and divergent nomenclature. Anyone who has tried to make an analysis of the charts kept by others knows how much he has to read into and between the lines; how often is it necessary to decide whether the doctor who wrote the history used the term "pigmented lesion" to mean black, while to others the same term may mean brown or red, whether a "large lesion" measures 1 cm. or 10 cm. in diameter, or whether a node listed as "nonpalpable" was correctely described as such. The list of variables could be extended, and the number of possible errors is compounded if those evaluating the charts hand them over to a third echelon for final analysis and interpretation. The more it is possible to reduce such variables, the more objective will be the end-results and conclusions.

In the present series all cases were preoperatively examined by the surgeon who also performed the operation and did the follow-up; preoperative sialangiography and

TABLE 1.—*Pathologic Classification of Parotid Tumors (218 Cases)*

Malignant Tumors (186 Cases)			
A. Primary (160 cases):		B. Secondary (26 cases):	
1. Mixed tumors	121	1. Metastatic	15
2. Mucoepidermoid tumors	29	2. Direct extension	11
3. Others	10		
Benign Tumors (32 Cases)			
1. Warthin tumors (papillary cystadenoma lymphomatosum)			18
2. Others			14

postoperative roentgen therapy as well were administered by one and the same radiologist, in close contact with the surgeon; there were frequent consultations with the pathologists in order to guarantee a uniform standard of classification; finally, all charts were evaluated and analyzed by the surgeon in charge. The material consisted of 218 cases of parotid tumors, all of them surgically excised and followed for periods of from 2 to 10 years. The method used in this study permits an answer to several of the problems concerning parotid tumors, while with regard to other questions a solution is at least suggested.

Of the 218 cases, 121 were mixed tumors, 29 mucoepidermoid tumors, and 18 Warthin tumors (Table 1). The remaining 50 cases comprised 19 different categories, and will therefore be disregarded in the statistical evaluation.

The age distribution of patients with mixed and mucoepidermoid tumors ranged from 16 to 82 (average 47) years; of those with Warthin tumor from 48 to 83 (average 65) years. The incidence of Warthin tumor was considerably higher in men, while mixed and mucoepidermoid tumors were much more frequent in women.

The location of the three common types of parotid tumors is presented in Table 2.

TABLE 2.—*Parotid Tumors, Location*

	Superficial Lobe			Deep Lobe
	Preauricular Area	Lower Pole	Pterygoid Extension	Includes Intraoral and Duct Lesions
Mixed tumors	14%	23%	33%	30%
Mucoepidermoid tumors	21%	17%	24%	38%
Warthin tumors	6%	72%	11%	11%

Warthin tumors were predominantly situated in the lower pole of the superficial lobe of the parotid, with only 11% occupying the deep lobe. Mixed and mucoepidermoid tumors were much less frequently encountered in the lower pole.

Diagnosis

In clinical examination the following criteria were used: appearance of the lesion; location; palpation to decide whether the swelling is free or attached to the overlying skin—this latter finding established the differential diagnosis from sebaceous cyst; palpation of surrounding areas, particularly the cervical lymph nodes, to detect local extension or metastatic spread. Palpation evaluated in the light of history suggests Warthin tumor, if the patient is an elderly man, the lesion is located at the lower pole of the parotid, is somewhat soft, and varies in size at different examinations.

Radiologic examination is of decided aid.[2] Sialangiography permits differentiation between inflammatory lesions and neoplasms, thus eliminating needless surgical explorations. It is furthermore useful in the presurgical localization of the tumor; sometimes the sialangiogram demonstrates involvement of the deep lobe where palpation had suggested a growth in the superficial lobe, thus alerting the surgeon to a more difficult dissection.

Rarely is it necessary to resort to biopsy before proceeding with definitive treatment. Silverman needle biopsy is hazardous since it may result in spread of tumor cells into the parotid gland or adjacent tissues; the field of operation would then have to include the entire needle track and often also neighboring structures, thus making it impossible in some instances to save the facial nerve. Pretreatment open biopsy through a small incision carries similar risks: it may endanger the facial nerve since the entire biopsy area will have to be encompassed as part of the subsequent operation. Frozen section biopsy is seldom indicated, but when it is performed everything should be held

in readiness for whatever surgical intervention may be required.[3]

Rationale of Management

Surgical excision was performed in all cases. The operation was standardized throughout, the only variant being the magnitude of the procedure, which depends on pathologic character and extent of the lesion. In the first place, a distinction must be made between encapsulated and infiltrating neoplasms. Most parotid tumors are well defined and limited by a so-called capsule. This "capsule" (directly related to the slow growth of the tumor) is only a condensation of fibrous stroma, in the interstices of which there are islands or clumps of tumor cells. The surgical objective is complete removal of the tumor together with its "capsule." To this end a shell of uninvolved parotid tissue surrounding the palpable mass must be excised. In the overwhelming number of cases this can be achieved by subtotal parotidectomy.

Total parotidectomy was never performed electively. Even when the size of the tumor made total or almost total parotidectomy mandatory, it was not done "en masse" as long as there remained a chance to spare the facial nerve.

The majority of infiltrating tumors are much more radiosensitive than the average "encapsulated" parotid tumor. Furthermore, a very high proportion (50% to 75%) metastasize at least to the regional lymph nodes. Unless one is prepared to perform total parotidectomy together with excision of adjacent condemned areas, the preferred management is radical neck dissection in continuity with removal of almost all the parotid tissue, while saving the facial nerve. By this plan the field of postoperative radiation is limited to the site of the primary lesion. The fact that the facial nerve has been preserved in no way diminishes the chances of roentgen therapy. Such a combination of modalities offers a better prospect for success than reliance on either one alone, as demonstrated in carefully selected cases.

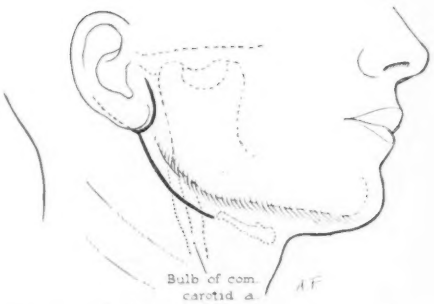


Fig. 1.—Illustrating the "Y" incision with the preauricular arm placed in a natural wrinkle very close to the tragus and the cervical arm paralleling an adjacent neck crease as it is extended to the tip of the hyoid. (From *California Med.* 86:223-228 [April] 1957, California Medical Association, San Francisco).

Radical neck dissection in continuity with extirpation of the primary tumor was performed (1) in the presence of clinically demonstrable involvement of one or more cervical lymph nodes; or (2) as part of the above-described combination of surgery and roentgen therapy, for infiltrating tumors. When the primary disease has been effectively controlled, spread to a cervical lymph node which would necessitate staged neck dissection is very rare. In the present series this complication occurred only once.

Technique

A "Y" skin incision with the "hub" immediately below the lobe of the ear was routinely used in this series (Fig. 1).

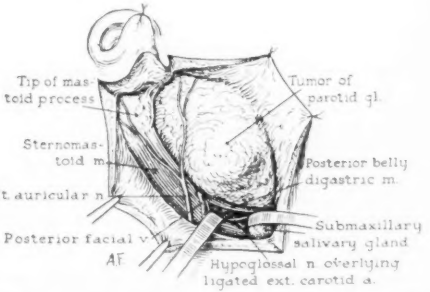


Fig. 2.—The anatomical landmarks displayed after elevation of the skin flaps. The external carotid artery may be ligated at this time just caudad to the posterior belly of the digastric muscle. (From *California Med.* 86:223-228 [April] 1957, California Medical Association, San Francisco.)

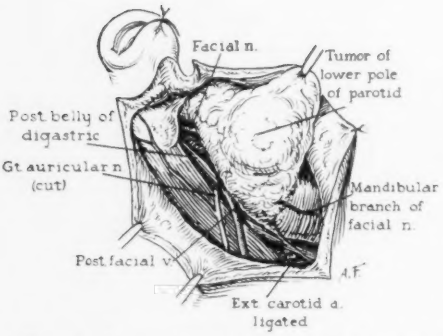


Fig. 3.—By tracing the posterior belly of the digastric muscle to its origin on the medial surface of the mastoid process, the main stem of the facial nerve is identified as it lies 1 to 2 cm. deeply medial to this point. When the volar surface of the tip of the index finger is placed on the medial surface of the mastoid, the nail will be in contact with the main stem of the facial nerve. (From *California Med.* 86:223-228 [April] 1957, California Medical Association, San Francisco.)

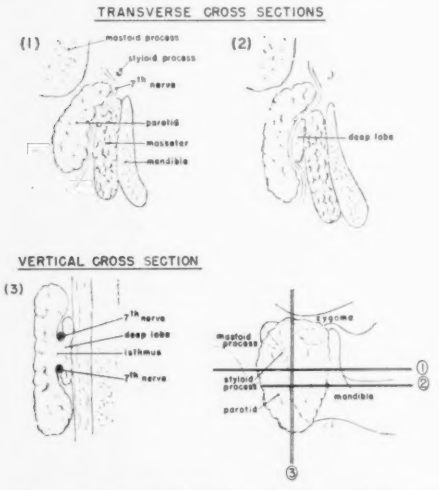


Fig. 4.—The relationship of the facial nerve to the lobes of the parotid gland.

The transverse section at level (1) is at a point above the isthmus of the parotid gland. It shows the facial nerve as it lies between the masseter muscle and the parotid. It is separated from both by a layer of fascia.

Transverse section (2) is at a lower level and passes through the isthmus of the parotid gland. Here the divided facial nerve is seen in contact with the deep and superficial lobes of the parotid gland. It is separated from both by an enveloping layer of fascia. It does not run through the substance of the parotid gland.

The vertical section through level (3) further illustrates the relationship of the divided facial nerve to the contiguous lobes of the parotid gland. Clinically this is well demonstrated by the ease with which the adjacent parotid lobes are spread and lifted away from the facial nerve and its branches.

The external carotid artery was ligated in continuity whenever it was felt that as bloodless a field as possible was necessary to avoid injury while locating the main stem of the facial nerve (Fig. 2). This was particularly helpful in cases of large or incarcerated tumors of the pterygoid extension of the superficial lobe, or in tumors of the deep lobe and in recurrent cases. The procedure can be performed through the same incision by extending the submandibular arm several centimeters toward the hyoid bone. The theoretical disadvantage of such ligation—that it would interfere with the clean performance of neck dissection later, if that were indicated—can be dismissed as being of little importance because the incidence of cervical node involvement in such cases is less than 1%.

In all cases the facial nerve was isolated at a point where its main stem emerges from the stylomastoid foramen (Figs. 3 and 4). None of the other approaches for isolation of branches of the facial nerve (Fig. 5) was utilized in this series. In previously untreated cases, regardless of the

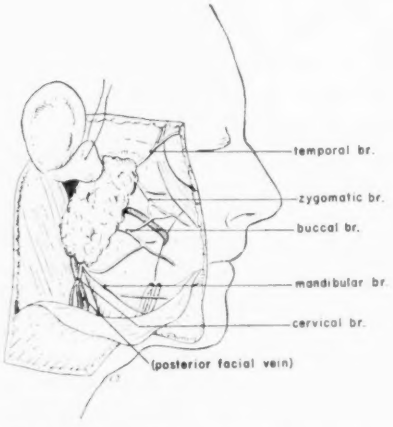


Fig. 5.—Diagrammatic presentation of the distal ramifications and anastomoses of the facial nerve, showing several of the relatively fixed points where the nerve branches can be located. These are where the cervical branch passes superficial to the posterior facial vein, the mandibular branch crosses the facial vessels, the buccal branch passes transversely or vertically over Stensen's duct, and the temporal branch passes deep to the fascia covering the zygomatic arch.

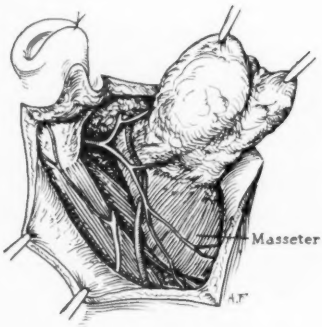


Fig. 6.—While dissecting out the branches of the facial nerve, the superficial lobe of the parotid is elevated with its contained tumor. Note that the posterior facial vein is usually deep to the facial nerve. (From *California Med.* 86:223-228 [April] 1957, California Medical Association, San Francisco.)

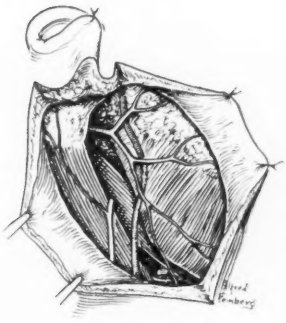


Fig. 7.—If the tumor is in the deep lobe, the superficial lobe must first be removed as shown in Figure 6, and then the diseased deep lobe may be removed from beneath the facial nerve by elevating or spreading the branches. (From *California Med.* 86:223-228 [April] 1957, California Medical Association, San Francisco.)

histologic diagnosis or size of the tumor, the facial nerve was never sacrificed (Figs. 6 and 7), except for instances of infiltrating nonencapsulated malignant tumor when the facial nerve was embedded within the substance of the spreading growth. Unless the surgeon is prepared to remove the adjacent condemned areas, such as the masseter muscle and possibly the auricle, the external auditory canal, and part of the mandible and mastoid, nothing is to be gained by sacrificing the facial nerve and leaving all or some of the involved contiguous structures in place. Simply removing the facial nerve together with the parotid gland makes the operation much easier, but does not improve the chances of complete extirpation of local disease.

When the preauricular area of the parotid gland is approached during dissection, a careful search is made for large tributaries of Stensen's duct; they are isolated and transected, and the cut distal ends tied with fine silk to reduce the likelihood of fistula formation. The incision is closed with an indwelling drain left at the lower end of the cervical arm. (Fig. 8).

Results

Subtotal parotidectomy is the procedure of choice, and was performed in 128 of the 168 cases (Table 3). In the remaining 40 cases total (or almost total) parotidectomy was required.

There was not a single instance of accidental injury to the main stem or to a major branch of the facial nerve. In six cases the main stem was deliberately transected—four times on account of presurgical paralysis, and twice because the nerve was intimately tangled in the crevices of huge recurrent tumors. The latter were actually multicentric foci of recurrent tumors which had become fused into conglomerate masses, trapping the nerve branches within the substance of the growth. Nothing similar was ever observed with "encapsulated" tumors, even large ones, which had not been previ-

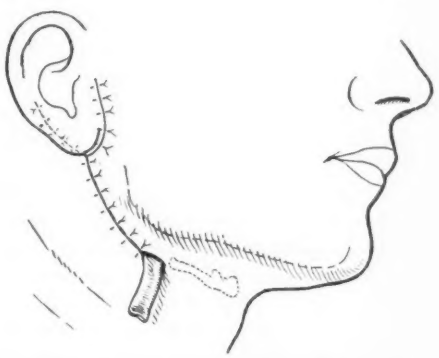


Fig. 8.—Closure, showing site of drain. (From *California Med.* 86:223-228 [April] 1957, California Medical Association, San Francisco.)

TABLE 3.—*Parotid Tumors, Kind of Operation*

	No. of Cases	Parotidectomy		Neck Dissection		Metastases			No. of Deaths
		Subtotal	Total	One-Stage	Two-Stages	Number of Cases with Metastases	To Cervical Nodes	Distant: Lung Bones	
Mixed tumors	121	98	23	2	1	3	1	3	2
Mucoepidermoid tumors	29	13	16	5	0	10	10	5	7
Warthin tumors	18	17	1	0	0	0	0	0	0
	168	128	40	7	1	13	11	8	9

ously disturbed. In cases not operated on the nerve branches are either stretched over the surface of the mass or—at worst—embedded in a crevice of the nodular tumor, from which they are separated by a layer of fascia, and can therefore easily be removed. In addition, smaller branches of the facial nerve (nine times buccal, seven times mandibular) were deliberately transected; the rationale for this decision was increased threat of further recurrences, inextricable adherence of the branches to diseased tissue, as well as their apparent functional unimportance. This latter point was borne out by the fact that in all but one instance complete function returned within two weeks to two years.[4]

Salivary fistula occurred in only 6.5% of cases of subtotal parotidectomy and in 8% of total parotidectomy; all cleared spontaneously in 15 to 87 days. Searching for major duct branches and tying them off with nonabsorbable fine silk may have been an important factor. The auriculotemporal syndrome developed in a surprisingly large number of cases (24%) between 2 and 48 months after the operation.

Metastases to the cervical lymph nodes occurred preoperatively in 10 cases of mucoepidermoid tumor (32%) and postoperatively in one case of mixed tumor (Table 3). Distant metastases to lungs and bones developed postoperatively in 2.5% of mixed tumors and in 16% of mucoepidermoid tumors. As expected, no metastases were observed in cases with Warthin tumor.

Nine patients died of their disease; the incidence was 1.6% in the group of mixed tumors, and 24% in mucoepidermoid tumors (Table 3). Death was due to distant metastases in six of the nine cases, and to local effects of the disease in the other three cases.[5]

Comment

The management of parotid tumors is based on a simplified pathologic classification.[6] Warthin tumors are the least controversial, and are commonly regarded as benign, even though they may be multicentric and bilateral. With regard to mixed and mucoepidermoid tumors the histologic criteria for a differentiation between benign, low-grade, and malignant growths are far from specific. From a clinical standpoint it is expedient to simplify the issue and to classify all these lesions as malignant. Mixed and mucospidermoid tumors seem to have a common genesis and differ only in their growth potential, with the mucoepidermoids representing the more malignant variety. In both groups the primary lesion may be "encapsulated" or infiltrating, and the disease may have progressed to metastasis or not. It is these two points which are decisive in planning the surgical management of parotid tumors of either type.

Perhaps the most important criterion for judging the effectiveness of any treatment method is the rate of recurrence at the primary site. McFarland[7] and others[8-13] showed that with simple enucleation, curettage, radiation, or a combination of these therapeutic measures, the local (parotid

TABLE 4.—*Recurrences at the Primary Site*

	No Previous Operation		Previous Operation	
	No. of Cases	Local Recurrence	No. of Cases	Local Recurrence
Mixed tumors	89	1%	32	18%
Mucoepidermoid tumors	14	None	15	13%

area) recurrence rate is from 15% to 65%. The higher figure is perhaps the more dependable, since it is based on a study of a large group of cases over a long period of time.[7] In the present series (Table 4) it was noted that the rate of local recurrence was very low in primary cases and much higher in those which had been previously operated. To be specific, no local recurrence took place among 14 primary mucoepidermoid tumors, while there was one recurrence among 89 primary mixed tumors; on the other hand, among mucoepidermoid tumors previously operated on the rate of local recurrence was 13%, among mixed tumors as high as 18%. In both categories surgical management was the same or even more extensive in the group of cases which had been previously operated on. It thus becomes evident that it is easier to achieve lasting control at first operation than at time of local recurrence.[14]

Total parotidectomy, often in combination with elective neck dissection, is frequently recommended as a method of management.[15] The rationale for such a policy is usually the assumed multicentricity of "encapsulated" parotid tumors in general. However, the incidence of multicentric growths is rare among "encapsulated" tumors which have not previously been treated.[16] Presumably the high percentage of multicentric "encapsulated" tumors encountered in the literature refers to cases which had been operated on, but where inadequate excision or enucleation had left remnants of the capsule with attached tumor cells in the perimeter of the tumor bed. From these multiple foci extension may take place into the field of previous operation, creating the wrong impression of original multicentricity and suggesting the need for a more radical technique.

Summary and Conclusions

The successful management of parotid tumors combines complete removal of the neoplasm with preservation of the facial nerve. It depends on two factors: a thorough understanding of the natural history of these tumors, and familiarity with the surgical anatomy of the area.

Clinically, parotid tumors may be divided into so-called "encapsulated" and infiltrating lesions. "Encapsulated" tumors make up about 95% of the cases and are managed by subtotal parotidectomy. Infiltrating tumors are treated like infiltrating cancer anywhere, namely by wide excision of the primary lesion in continuity with the immediate drainage area, by means of a radical neck dissection, with or without postoperative radiation.

The series included 218 parotid neoplasms, of which 150 were either mixed or mucoepidermoid tumors. In this latter group there were 103 cases which had not been previously operated on, with only one local recurrence, amounting to less than 1%.

A technique is described by which this result was achieved without sacrificing the facial nerve. In the entire series there did not occur a single instance of accidental injury to the main stem or one of the major branches of the facial nerve.

300 S. Beverly Drive.

REFERENCES

1. Perzik, S. L.: Parotid Tumor Operations: The Case Against Enucleation, California Med. 85:26-29 (July) 1956.

2. Einstein, R. A. J., and Perzik, S. L.: Parotid Sialangiography, California Med., to be published.

3. Lucas, J. C.; Newman, W., and Klopp, C. T.: Significances of a Lump in the Parotid Gland, Postgrad. Med. 19:350-353 (April) 1956.

4. Perzik, S. L.: Facial Nerve Paresis in Parotid Surgery, Surgery 36:751-761 (Oct.) 1954.

5. Perzik, S. L.: End Results in Parotid Tumors, California Med. 86:223-228 (April) 1957.

6. Perzik, S. L.: A Clinicopathologic Correlation of Parotid Tumors with Therapeutic Applications, J. Internat. Coll. Surg. 18:479-482 (Oct.) 1952.

7. McFarland, J.: The Histopathologic Prognosis of Salivary Gland Mixed Tumors, Am. J. Med. Sc. 203:502-519 (April) 1942.

8. Ahlbom, H. E.: Mucus- and Salivary-Gland Tumours: Clinical Study with Special Reference to Radiotherapy, Acta Radiol. Supp. 23, pp. 1-452, 1935.

9. Bailey, H.: Parotidectomy: Indications and Results, Brit. M. J. 1:404-407 (March 29) 1947.

10. Devine, K. D.; Figi, F. A., and Beahrs, O. H.: Management of Recurring Parotid Tumors, A. M. A. Arch. Otolaryng. 63:552-555 (June) 1956.

11. Martin, H.: The Operative Removal of Tumors of the Parotid Salivary Gland, Surgery 31: 670-682 (May) 1952.

12. Stewart, F. W.; Foote, F. W., and Becker, W. F.: Muco-Epidermoid Tumors of Salivary Glands, Ann. Surg. 122:820-844 (Nov.) 1945.

13. Swinton, N. W., and Warren, S.: Salivary Gland Tumors, Surg., Gynec. & Obst. 67:424-435 (Oct.) 1938.

14. Lyle, F. M.: Surgical Consideration of Parotid Tumors, Am. J. Surg. 91:332-338 (March) 1956.

15. Brintnall, E. S.; Tidrick, R. T., and Huffman, W. C.: A Simplified and Rapid Anatomical Approach to Parotidectomy, A. M. A. Arch. Surg. 71:331-336 (Sept.) 1955.

16. Frazell, E. L.: Clinical Aspects of Tumors of Major Salivary Glands, Cancer 7:637-659 (July) 1954.

Rhinology: 1900 to 1910

A Brief Survey and a Bibliography

MAURICE H. COTTLE, M.D., Chicago

. . . the space of one man's life is insufficient for him to know anything but the rudiments of our art in many branches, and be, at the same time, in a position to advance in any degree the boundaries of its smallest province . . . we may pause (therefore) I trust (for) a just consideration and appreciation of the labor of our predecessors. Our knowledge has been built up . . . not by the mushroom activity of any one period, or of any one school of medicine, or by the premature birth of an idea or theory, but by the painstaking, laborious exertions of many generations of earnest men, working, for the most part, without expectation or perhaps desire; certainly without the attainment of those rewards, by which not only the layman, but alas, even the average member of our own art, measures what he calls success.

These words, written in 1902 by Jonathan Wright, concluded a lengthy survey of the story of our art covering a period of 5000 years.

The poetry editor John Ciardi,* in a recent essay emphasized that no great progress is made without a sense of the past as well as of the present. He says, "No painter can produce a good canvas without a broad knowledge of what has been painted before him. No architect can plan a meaningful building except as he has pondered the architecture of the past, and no writer can produce good writing without a sure sense of what has been accomplished in the past within his form."

From the history of rhinology, I have selected the first decade of the 20th century for reviewing because it seems to me to be a "golden period," during which the accomplishments of the past blossomed into their fullest glory and new seeds were developed and sown for flowering in the future. The rhinologic topics receiving much attention during this decade were septum surgery, rhinoplasty, anatomy, and physiology.

In the preceding years, operations on the septum were gross, destructive, crushing procedures involving mass movement and mutilation of bone, cartilage, and mucosa alike, which depended upon nature's healing properties for their justification and toleration. The crushing operation of Asch, the V-shaped incisions and excisions of Kyle, and the flap operations of Gleason were among the better known and most frequently used. However, Ingals in 1882, Krieg in 1889, and Boenninghaus in 1899 were already emphasizing the need of careful submucosal manipulation of the hard parts of the septum. Their work, together with the works of others, made possible the beautiful and significant presentations by Freer. The English version of Killian's classic article, "The Submucous Window Resection of the Nasal Septum," by E. Edwin Foster of New Bedford, Mass., was published in the *Annals of Otology, Rhinology and Laryngology,* 1905. Here are some of Killian's conclusions: "The essential feature of the technique is that I make by means of my long speculum a medial space in which I can as easily operate as in the inside of either nasal cavity . . . the experienced operator can rapidly remove large pieces. I can perform the whole operation in 20 minutes . . .," and his last conclusion is, "I have never had more enjoyment from any other small operation than

Submitted for publication Aug. 20, 1957.

A complete bibliography will appear with the reprints.

From the Chicago Medical School and the Cook County Hospital.

Read at the 26th Annual Mid-Winter Clinical Convention, Research Study Club, Los Angeles, Jan. 14, 1957.

* Saturday Review of Literature, Dec. 15, 1956, p. 7.

from the described septum operation, and can most enthusiastically recommend it."

In the same issue of this journal, William Lincoln Ballenger first described the use of his swivel knife, which, he wrote "enables me to perform the operation in from 5 to 20 minutes . . . with the swivel knife the cartilage can be removed in 5 to 30 seconds." He described the use of a great many instruments, saying that "more instruments means more lacerations." His final justification for his presentation can be summed up in the following quotation: "After a reasonable experience it seems probable that the great majority of operations need not require more than 10 minutes. Killian claims an average of 20 minutes, while Freer by his methods claims an average of 45 minutes." This stress upon the time factor is quite revealing.

Important contributions were made by Hajek, Menzel, Leon E. White, Weil, Zarniko, Müller, Spratt, Beck, McCaw, Carter, MacCoy, Richardson, Sluder, and most of these were carefully studied by Freer in the course of this decade. He quoted from many in his articles, and apparently gave them serious consideration in estimating his own contributions. His conclusions from the article in 1905 should, in part at least, be restated and reevaluated:

1. The appearance of the deflections does not divide them into two great groups with distinct aspects, one obviously traumatic, and the other due to faulty growth as is taught by Killian . . this is merely confusing. . .

2. The window resection is adapted to children, but the chance of a possible recurrence from the effects of growth demands complete removal of the vestiges of deflection.

3. The firmness . . . of the septum is completely reproduced in the window after the resection (!!!).

4. Cases seen two and a half years after the operation show permanency of the result. (Note: 20 years is the time for evaluating permanency.)

5. Though the author (Freer) has never seen a case of sinking in of the nasal bridge after the nasal resection, Müller's and Menzel's warning should be heeded, to retain a strip of the cartilage of the septum under the lateral cartilages of the external nose. (Note: Also advised by Hajek.)

6. Lower portion of the quadrangular cartilage . . . may be resected without fear . . . (Note: Surely not so.)

7. The recumbent position of the patient is best for the operation, except in operating on the nasal floor.

8. The Kirstein light is the most suitable one.

9. The use of powdered cocaine. . .

10, 11, 12, and 13. Instruments.

14. Even if strictly subperichondrial, the elevation of the mucosa is apt to encounter adherent places that need separation by keen dissection. (This cannot be overstressed.)

15. The cutting out of denuded cartilage in one piece is the easiest part of my operation. (Note: When it is that easy, removal often can be avoided.)

16. . . . Neither cartilage nor bone should ever be broken, twisted, or torn from its attachment, but should always be cleanly cut away.

17. Instruments.

18. There is a tendency to hasty and incomplete removal of the bony part of the deflection.

19. Sewing is needless.

20. Packing.

21. Considering the many difficult bony resections met with, the author (Freer) does not think that the operation can be done in 20 minutes, the estimated time of Killian. (Note: Much more difficult by far is the freeing of the multifractured and distorted cartilaginous deflections, especially those mixed with bony deformities and scars in the anterior inferior part of the nasal septum.)

Freer was nurtured in the scientific atmosphere of Chicago which revolved primarily around Ingals. Freer's talented and industrious work affected all his colleagues. His accomplishments guided the younger men about him, but none were so effectively influenced as was Samuel Salinger, until recently also of Chicago, who I think became the direct successor of Freer and Ingals and brought to our times the finest scientific surveys of rhinologic literature and most significant contributions to the surgical advancement of our specialty.

An outstanding contribution was written by Dr. Sidney Yankauer of New York City (1906). He referred frequently to the work of Freer as well as many other contemporaries. He was especially aware of the difficulty of separating the anterior inferior border of the septal cartilage from the underlying bone. He described minutely the anatomy of this area and pointed out the

need for sharp severance of the soft tissues lying between the two structures before safe and adequate separation of the nasal mucous membrane on both sides could be accomplished.

It was pointed out by John N. McKenzie in the *New York Medical Journal*, 1905, that the craze for nasal operations had caused the pendulum to swing too far from rational boundaries: that the amount of reckless surgery in the nasal passages would never be known.

In the *Laryngoscope* of 1906 in a splendid critical and long review, John R. Winslow of Baltimore concluded that there is no single method suitable for every variety of deformity but all methods give good results in certain cases. It seemed to him that indications for the selection of each particular operation had not been sufficiently defined. He justly pointed out that the lack of detail in recorded cases would seem to indicate insufficient study; further, that the older methods had not met the requirements and had not proved entirely satisfactory. He made many significant observations, viz., "Inspection under cocaine anesthesia is essential to diagnosis, but inspection alone often yields erroneous impressions. The graduated probe is often the requisite to determine the extent of the deformity, the consistence of projections and the depth of concavity. The object of operation is the restoration of absolute symmetry." He concluded that conservatism and judgment in selection of cases are indispensable. He quoted Semon, "The magnitude of operations must not exceed the importance of the symptoms." He referred to the suggestion of C. W. Richardson of Washington that leaving splints in the nose for a period of 7 to 10 days after the operation simplified the after treatment. He quoted Roe, Bosworth, Hajek, Killian, Menzel, Janson, Freer, and Ballenger. He believed that the septum is not a mere partition but a support of the external nose. He described the operations of Harrison Allen in which the maxillary crest is severed by means of a small chisel placed beneath the upper lip, and the more extensive but similar operation by Lowe. He concluded that a knowledge of all methods is needed and that the older methods should be modernized if necessary, rather than abandoned, and that submucous operations of the nasal septum belong to the most difficult of operations, that they are indispensable and should be mastered by everyone who aspires to the title of the modern nasal surgeon.

In his presidential address to the American Laryngological, Rhinological, and Otological Society, 1904, Frederick C. Cobb of Boston said, "How numerous have been the operations devised upon the nasal septum, but how many of their originators will say in a medical meeting today that their methods have been improved upon."

Goodale and others in the Boston area, with the influence of the already familar work of Roe and Weir, probably created the groundwork for the development of interests in rhinology which were presented to the profession in the great works of Mosher. His (Mosher's) outstanding contribution at this period entitled "The Premaxillary Wings and Divisions of the Septum," presented at the 13th Annual Meeting of the American Laryngological, Rhinological and Otological Society in 1907, stressed the need for the study of the anatomy of the septum, the development of the premaxillae, and their relationship to the growth of the incisor teeth. Mosher described the relationship between deviated septa and delayed and irregular dentition; he illustrated examples of the deviation of the septum found in the dissecting room and showed some to be due to displacement of the premaxillary wings.

He discussed the relationship between the deformities of the palate and deviations of the septum and suggested that trauma is a cause of these abnormalities. Mosher was also concerned with the correction of old fractures of the nasal bones and knew that the usual methods of treatment were inadequate. His beautiful drawings show very clearly his "seeing through" a nasal de-

formity and recognizing its underlying concomitant septal derangements. He described a direct method of correcting lateral deformity of the nasal bones, employing external osteotomies, a method which with slight modification is still being used extensively today. He described in great detail the anatomical changes in nasal fractures in the external portion of the nose and of the septum and discussed many details for mobilizing the parts involved, emphasizing the need of handling the ascending process of the superior maxilla and also the deformities of the septum at one and the same time. He wrote that in most old fractures of the nose there is not only deformity of the nasal bones but also of the lower half of the nose or the cartilaginous parts. He added, "The anterior edge of the quadrangular cartilage is responsible for the support and so for the shape of the lower half of the bridge." Further he says, "In order to get the anterior edge of the quadrangular cartilage into the middle line, it is necessary to free it from the skin which lies over it and to replace its base." He tried to accomplish this by incisions through the septum and breaking the crests and forcing the septum into a new position and holding it there by considerable packing. He knew that this was not satisfactory very frequently and was prepared for secondary operations and concluded that the correction of the deformity of the cartilage is much harder and less sturdy than correction of the deformity of the nasal bones. Mosher's description and drawings of the anatomy of the septum of the infant and of the adult, with his emphasis of the part played by the premaxillae and their relationship to the quadrangular cartilage, the vomer, and the maxillary bone and his correlation of the growth of these structures with the growth of the palate, the maxillae, and the upper dental arch and dentition, will stand as a monumental contribution to the science of rhinology and as an inspired stimulus to all who would study this subject today. But Mosher in his publications after 1910 made no further reference to this work which he had so brilliantly inaugurated. And, I might add, as far as I know, further work by others along the lines of his original investigations was not done for several decades.

Chevalier Jackson in 1902 presented a discussion of why corrective operations of deviations of the nasal septum often failed. His article is splendidly written and well illustrated. He observed the relationship of sleep and rest to the position of the head and to the turgescence of the inferior turbinates, but was, in my opinion, incorrect in his conclusions that the swelling of the inferior turbinate, especially during sleep, pushed the septum and caused deviations. His conclusion on the inadequacy of the septum operation was that the surgeon had failed to remove a portion or all of the inferior turbinate on the concave side of the septum. He finished his article with this exhortation: "Let the skeptic not theorize—go cut out the turbinates." Yet Goodale in 1896 had emphasized the variations of the route of inspired air due to the shape and size of the vestibule, the inferior and middle turbinates, and the septum, and Ziem's article on the physiology of the inferior turbinate was soon to be published. Lothrop, Hartz, and others also had called attention to the functional importance of the inferior turbinates, and Courtade's graphic measurement of the permeability of the nasal fossae had appeared in 1902. Chevalier Jackson's conclusion after observing for two or three years his patients who, in addition to septal resection, had had some partial or total inferior turbinectomies, was that his patients had benefited greatly from the surgery. It may be repeated, I feel, that a careful scrutiny of nasal surgery a decade or two after it is performed is necessary before dependable scientific evaluations can be made.

The beginnings of rhinoplasty in early times and its gradual slow growth is fairly well known. A few brief remarks on some of the highlights may be made.

The Edwin Smyth Surgical Papyrus, 3000 B. C., mentions the treatment of nasal

fractures and the use of pressure dressings postoperatively. In 600 B. C. the time of Susruta, major restorations of the nose, repair of the nasal tip from cheek flaps and forehead flaps were done. The great Tagliacozzi brought to world notice the work of the Brancas and his own epochal efforts. The great master plastic surgeon of the 19th century, Diffenbach, was responsible for arousing further interest in partial and total reconstruction of the nose. He also straightened "twisted" noses. In 1887 John Roe of Rochester, N. Y., probably was the first to introduce intranasal incisions for rhinoplasty. He was concerned with the reduction of the bulbous tip and used cross hatching of nasal cartilages for mobilizing them and also was familiar with external molded splints. He might probably be the one to be called the originator of corrective rhinoplasty. He did hump removals; he used cocaine for local anesthesia. About the same time Robert Weir of New York was concerned with what may be termed minor rhinoplastic procedures and probably was the first to introduce the narrowing of the wide nose by means of excisions at the base of the nasal alae. He performed lateral osteotomies intranasally and used steel needles through the nose to hold the mobilized bones in place. He also reported the implantation in the nose of the breast bone of a young duck. Czerny in 1895 cut the superior portions of the upper lateral cartilages, bent them anteriorly to their attachments to fill in a depression in the dorsum of the nose—a procedure which was reported again 50 years later. As early as 1896 autogenous bone from the tibia was transplanted by Israel. He also transplanted autogenous cartilage from the ribs.

In 1902 Koenig used the combined free transplant from the ear to correct minor deformities of the nose, a method to be reviewed two generations later.

The great Jacques Joseph of Berlin at the beginning of this century was doing hump removal by excising V-shaped segments of skin, bone, and cartilage and was removing triangular wedges at the caudal end of the septum for shortening of the nose. In 1904 he reported on hump removal done completely intranasally through intercartilaginous incisions. He introduced the use of his right-angled saws for the intranasal lateral osteotomy, but perhaps one of his greatest contributions was his emphasis on the need of photographs and casts and good written records for both study and other scientific purposes. One of his articles in 1902 concludes with this very revealing observation, "Depressed unhappy people became gay, lively and interested in living after cosmetic surgery." This article concluded with the sentence telling of a patient who after saying goodby to him, turned back from the doorway and said, "You don't know how lucky and happy I am—nobody stares at me any more." At this time, the immortal Sigmund Freud was at the zenith of his creative achievements, but the works of these two great masters of medicine required two generations of further development before amalgamation of their experiences into a common arena of understanding could occur.

Joseph was conscious of the need for anatomical studies of the nasal septum. His concept of shortening the nose by means of shortening the septum led him to investigations concerning the septum even though he was not a rhinologist.

Transplants of autogenous, isogenous, and heterogenous bone were well known and used by Joseph and by many others of this period. In 1905 Welty reported a case of transplantation of the patient's tibia to the nose. He writes that he was about to report his operation as an original one, but on looking up the literature he had found that the identical operation had been satisfactorily performed by many before. Five years later the sacrifice of the second finger of the left hand as a nasal implant and graft was still being enthusiastically reported (Finney; McGraw). Goodale in 1901, however, used autogenous septum cartilage as implants for the correction of nasal deformities. He was

familiar with lateral osteotomies done intranasally and without completely sawing through the bone. He was one of the first to mobilize the nasal bones freely and to use external splints.

This first decade of the 20th century was also one in which great contributions to the anatomy and embryology of our specialty were being made and outstanding work previously published was being reintroduced to the profession. The rhinologist's own acute interest in septum, nasal, and sinus surgery was the magnet which attracted him to these basic sciences. Anatomical contributions of Zuckerkandl were outstanding. This great master prepared and studied sections of the head and nose, and his research in comparative anatomy of sinuses and other nasal structures gave us many fundamental concepts. He described the paraseptal cartilages, which of course led to the suggestion of their involvement in septal deformities. Onodi in Europe and Loeb in America made telling contributions to the anatomical knowledge of the nose and sinuses. Sudler from Johns Hopkins, Talbot, and Fawcett, among others, were vitalizing embryological investigations which were of important interest to rhinologists. The studies in physiology of the nose by Zwaardemaker and Mink provided in this decade all that was necessary for the development of extensive interest in nasal physiology and its relationship to the treatment of nasal diseases. In this country Hartz wrote about the peripheral stimulation of the nasal sensory nerves producing dilatation of nasal blood vessels while producing vascular contraction in other parts of the body. Anderson did research on dogs and found that occluding the nares resulted in loss of hair of the animal and that long after the stenosis was relieved the life span of the progeny was markedly reduced.

All of the foregoing and much more besides was going on 50 years ago, and yet on serious reflection all still seems important today. In the interim the newly developing advances in sinus surgery, otology, laryngology, and endoscopy absorbed the time and attention of the members of our specialty. Others took over much nasal surgery which the rhinologists had first explored and then partially abandoned. It is to the everlasting credit of Dr. Samuel Fomon that the otolaryngologist was made aware of his great heritage and of his need to concern himself again with the problem of nasal corrective surgery.

Perhaps the greatest lesson one can learn from this outstanding decade in rhinology is that advancements in medicine go on. Some of the past must be integrated into the needs and the knowledge of the present. Reevaluation and reassessing of the efforts of others and of ourselves is essential if the striving for the good and the true be our objective. Medicine, like all other forms of human activity, moves and changes, is dynamic, not static, and progresses ever onward toward a goal which may never be reached, but always is worth striving for.

As Jonathan Wright said, "Very frequently a new triumph of dexterity of invention in surgery leads to the erroneous assumption that a new era in the specialty has been inaugurated." We have seen many things considered original and new today which have been discovered or known long ago. Sir William Osler† reminded us, "The past is always with us, never to be escaped; it alone is enduring, but amidst the changes and chances which succeed one another so rapidly in this life, we are apt to live too much for the present and too much in the future. . . . It is good to hark back to the olden days and gratefully to recall the men whose labours in the past have made the present possible."

It is conceivable that our successors some time hence will again enjoy surveying the record of rhinology during this first decade of our century and I trust that it would not be too daring to hope that they may discover that a second "golden" decade flourished about 50 years later.

†Osler, W.: Aequanimitas; with other Addresses, Ed. 3, Philadelphia, The Blakiston Company, 1932, pp. 8-9.

Summary

Outstanding and significant contributions to nasal septum surgery, corrective nasal surgery, anatomy, and physiology were made during the first decade of the 20th century. The record of these achievements can be read with great profit today and is an inspiring stimulus for those working in these fields.

30 N. Michigan Ave.

Sinusitis and Nonspecific Endogenous Ocular Inflammation

JOSEPH LUBART, M.D., New York

Tables covering the causes of blindness, compiled by the Commission for the Blind of the New York State Department of Social Welfare, Jan. 13, 1955, show that in more than 40% of the cases covered the cause was "unknown to science." In a report by Lancaster and Foote[1] published in *The Journal of the American Medical Association*, our blind population was placed at 260,000. They also estimated that 22,000 persons have their visual acuity reduced to 20/200 or less each year. Physicians attending the Eighth Annual Assembly of the American Academy of General Practice heard that approximately 27,500 Americans became blind during 1955—a record-breaking figure. Another 1,000,000 persons are estimated to be blind in one eye. Studies show that about 12% of blindness is due to uveitis, and the actual percentage must be greater when one considers the cases diagnosed clinically as primary glaucoma, which on pathological examination, after enucleation, are found to be affected by chronic uveitis with secondary glaucoma.

The treatment of uveitis has gone through many phases and differs in different countries. Syphilis was considered the cause in France and antisyphilitic treatment was used even when little evidence of syphilis existed. The widespread use of penicillin has largely eliminated this disease. Treatment for tuberculosis was the vogue in Germany in patients afflicted with uveitis. Focal infection was considered the etiological cause of uveitis in England, and teeth, tonsils, sinuses, prostate, gallbladder, and even the appendix was at one time or other held to blame. In this country opinion varied, and some authorities used antisyphilitic and tuberculin therapy, whereas others removed or treated foci of infection. With the advent of the chemobiotics, especially since the addition of the corticosteroids to our armamentarium, treatment with one or other of these drugs has overshadowed all other approaches to the problem. It is becoming increasingly evident, however, that these new drugs, although often dramatic in their influence on the acute or subacute phase of disease, have little or no effect once the inflammation has reached the chronic form. Unduly prolonged therapy of any sort, if not productive, carries with it the penalty of irreversible changes in the uveal tissues.

There is a great deal of evidence of an intimate relationship between the orbit and the paranasal sinuses. For example, catarrhal conjunctivitis accompanies the coryza in nearly every case of measles. Hay fever, a recurring seasonal condition, and vasomotor rhinitis, which is all-seasonal, are accompanied by congestion of the conjunctiva. It has been noted that patients affected with marked photophobia will have paroxysms of sneezing when exposed to bright light and that mechanical irritation of the nasal mucosa can produce changes in the caliber of the retinal vessels on direct observation with the ophthalmoscope.[2] There are anatomical facts which establish a close relationship between the eye and the paranasal sinuses. The orbit lies imbedded in the cranial sinuses. Two-thirds and in some cases three-quarters of the circumference of the orbital sac is contiguous with the sinuses. According to Schaeffer,[3] the osseous boundaries of the ethmoid capsule are frequently defective, especially in the thin

Submitted for publication July 8, 1957.

Read at the Sixth International Congress of Otolaryngology, Washington, D. C., May 8, 1957.

orbital plate. The osseous wall between some of the anterior ethmoidal cells and the lacrimal sac is frequently deficient, and as a result the mucosa of the cells comes into actual contact with the lacrimal sac. Other studies of Schaeffer[4] have shown extreme thinning and dehisences in the osseous wall of the frontal and sphenoid sinuses, with the possibility of extremely close association of the optic and other orbital nerves with the posterior sinuses, especially with widespread pneumatization. Pathways connecting the eye and the nose by means of vascular and lymphatic channels and bone-marrow spaces have been shown to exist. Extension of the inflammation along the nerve sheaths has also been demonstrated as a possibility. The anatomical relations are such that disease of the paranasal sinuses could affect the eye, either by direct extension of the inflammation to the orbital tissues by contiguity or by the pressure of the inflammatory swelling of the mucosa lining the foramina of the many nerves and blood vessels which are common to both structures.

It is difficult in most cases of sino-ocular inflammation to establish cause and effect because chronic sinusitis of varying degree has a very widespread incidence, whereas orbital disease is selective, and the orbit, being a highly specialized organ, is extremely sensitive to inflammatory change, while the nose is a relatively simple structure, and, although exposed to constant infection and reinfection, is able to function after a manner even under the most adverse conditions. It is well known that the acute and subacute phases of sinus inflammation are either self-limiting or can be controlled in most cases by proper treatment. The problem lies in the long-standing type of case, where fibrous adhesions and invasion by the infection of the underlying structures causes glandular degeneration or hyperplasia, with cysts, abscess formation in various stages, chronic periosteitis, and finally osteitis. The newly discovered forms of medical therapy, so useful in localizing the infection in the earlier types of sinusitis, are useless here, as tissues where such changes have taken place can never be restored to normal. Further, although acute sinusitis does not lack for signs and symptoms, the chronic phase, which follows one or more attacks of the acute form, may have few manifestations. In latent sinusitis, all evidence of activity has long disappeared, but the sinuses continue to harbor walled-off areas of suppuration which under favorable circumstances may act as foci of infection.

Deep-seated infection in the sinuses is often difficult to demonstrate. It is, as previously mentioned, frequently asymptomatic. The x-rays are not always helpful; if the radiographs are positive, there is no sure way of determining whether the disease is past or present. Negative x-rays do not always rule out the presence of sinus disease. Johnson,[5] among others, in cases with positive clinical and negative x-ray evidence found, in a high percentage, that the maxillary sinus contained thick mucus and mucopurulent clots and in some cases frankly purulent material. More recently Semenov[6] stated, "A latent sinusitis may have nothing more to offer in the way of symptoms and signs than a history of nasal obstruction. The radiographic findings may be minimal, and yet when such a cavity is irrigated and a relatively small amount of mucopus is removed, the clinical improvement is striking." These statements were made with reference to the maxillary sinuses, which are of relatively simple construction. How much more complex is the problem as regards the entire cranial sinus labyrinth. Radiographs of the ethmoids can be especially deceptive. Ethmoid cells may involve the orbital roof or floor, or migrate into the perisphenoid or episphenoid regions, where the radiographic appearance is modified by adjacent soft tissues and bone. Clear ethmoids may be due to absorption of the intercellular septa rather than absence of disease. Variation in the thickness of the cortex of the anterior wall of the frontal sinus often makes detection of inflammatory

change difficult, while, because of its location, visualization of the sphenoid is frequently interfered with by adjacent dense structures. In the well-pneumatized sinuses, cells from the anterior and posterior ethmoidal groups may grow into the middle turbinate, the agger nasi cells, and uncinate process. Septa, more or less complete, recesses, and diverticulae may be found in the frontal, sphenoid, and maxillary sinuses. In the large sinuses, extension of pneumatization into the pterygoid process, greater or smaller wings of the sphenoid, the dorsum sellae and posterior clinoids, and even extention into the rostum and the nasal septum has been reported. Such extensions, where present, increase the likelihood of interference with sinus drainage and ventilation.

Study of the bacteriology of the nose is likewise not very helpful as deep-seated diseased sinus cells are usually completely cut off from the nasal cavity and can only be entered after surgical intervention. Furthermore, in many cases of uveitis the inflammation appears to be of an allergic type, but, while allergy may be an important factor in acute sinusitis, in the latent form it becomes a secondary influence. It goes without saying, however, that when allergies are found, they should be eliminated, or where this is impossible the patient should be immunized against them. The observations of Kern [7] in this connection are very illustrative: "If we examine pollen cases during the pollen season there is plenty of evidence of apparent abnormality in the antra on transillumination and x-ray. Yet if we examine these patients just before or a short time after the season, we find no more sinus involvement than in a group of normal controls. In fact, in our experience, the x-ray evidence shows their sinuses to be in better condition than the average population in our city. However, just as soon as the allergic state is perennial, that is, a vasomotor rhinitis or asthma, and it matters not which, the incidence of the sinus involvement is high: 89% in our vasomotor cases and 87.5% in four hundred asthmatics." In other words, the difference between seasonal and perennial rhinitis may be simply a difference in degree of sinus involvement; that as the sinusitis becomes more chronic, irreversible changes with interference in sinus drainage caused by repeated infections and adhesions, continue the inflammatory reaction throughout the year. Hansen-Pruss [8] talks about bacterial allergy but arrives at the same conclusion. He stated: "It is obvious that a large number of allergic individuals develop sooner or later a hypersensitivity to bacterial products. It follows, therefore, that proper surgical procedures directed to remove the foci of infection, in this instance, otolaryngological operations, are essential to solve the problem of these patients." Castroviejo,[9] a pioneer is corneal surgery, says, "In the course of my experience in keratoplasty, I have found the cornea to be an extremely sensitive barometer of the presence of infection, past or present, in the paranasal sinuses."

Focal infection for many years was thought to be a factor in the transmission of infection from one organ to another; several theories are used to explain how septic foci which frequently are pyogenic cause lesions in the eye which are plastic. It is stated that the presence of organisms in the blood (i. e., chronic septicemia) is not essential to the production of ocular infection but that chronic toxemia (i. e., circulating toxins) is. It is further proposed that septic foci elsewhere occasionally release a spurt of micro-organisms and that these, settling in the eye, cause inflammatory reaction, attenuation of the infecting agent taking place during passage through the blood stream. Bacterial allergy, already mentioned and as first developed with reference to the tubercle bacillus, is favored by some as the means of transmission of the toxic effects from one organ to another, while the theory of selective affinity, as formulated by Rosenow,[10] is used to explain why the ocular tissues alone are influenced by such bacterial toxins. Woods [11] uses desensitization with specific Streptococcus vaccine in his cases

of uveitis. I use autogenous vaccine prepared from material removed at operation to supplement sinus surgery when indicated, because the term complete sinus operation is more relative than real, since we have bone, scar tissue, and regenerative mucosa with bacteria present which may continue the infection, and it should be remembered that when sensitized tissue is present the gross extent of the pathology in any focus is irrelevant.

Direct proof of any of these avenues of spread of disease from the paranasal sinuses to the eye is difficult to obtain. Our clinical experience would lead us to believe that the mechanism of transmission in sinoorbital inflammation differs in each case and may involve one or more of the avenues mentioned above. In the present state of our knowledge one can only conjecture as to the actual method of transmission, and clinical proof is all that can be offered.

Report of Cases

Hemorrhagic Retinitis

CASE 1.—A man aged 21 years. Complete loss of vision in the left eye following three-year history of hemorrhagic retinitis complicated by retinal detachment and cataract. Present examination shows similar condition in the right eye with turbidity of the vitreous and blurring present for the past month. The vision in the right eye is reduced to 20/100. Repeated physical examinations and laboratory tests were negative except for clouding of the left ethmoid sinus. Aug. 7, 1951, bilateral external frontoethmosphenoidectomy was done. Within one month after surgery the retinitis had cleared, and vision in the right eye returned to 20/20. There have been no further retinal hemorrhages.

Comment: With irreversible changes causing total loss of vision in the left eye and the right eye apparently following a similar pattern, it was felt that half measures, if not successful, might jeopardize any chance of saving the right eye. The extreme youth of the patient, the absence of blood dyscrasias, the failure of all other treatment, and the speed with which the right eye responded to nasal surgery could leave little doubt but that the original cause of the trouble lay in the paranasal sinuses.

Uveitis

CASE 2.—A man aged 34 years. Six months' history during which the patient, a physician, had lost all practical vision. Examination on June 30, 1949, revealed bilateral uveitis with numerous vitreous opacities, and a few lens opacities beginning to appear in O. D.; cataract formation and complete posterior synechia O. S. After pansinusectomy the eyes became quiet, and the vision in the right eye gradually improved until it was 20/30 after several months and 20/20 at a later date. Aug. 6, 1954, right eye developed a retinal detachment, which was successfully treated, and the vision, which had become greatly diminished, was restored to 20/20 again.

Comment: This was a case of rapidly progressing uveitis, and during the six months from the onset of the uveal inflammation the patient had spent most of his time in several hospitals where all the known examinations and treatments were used, but the condition continued to degenerate. Fortunately the changes in the right eye were reversible and following corrective surgery restoration of the vision was complete.

CASE 3.—A woman aged 30. Had spells of inflammation in both eyes for 18 years. First seen Nov. 1, 1948. O. D., no vision for past several months because of uveitis; O. S. early uveitis. Several intranasal surgical procedures were done, with varying intervals of remission, which, however, were followed by attacks of increasing severity and duration. Oct. 27, 1951, a left external ethmoidectomy was performed. After this the uveitis subsided. Vision O. S. improved seven lines on chart, to where it had been in 1946, at 20/20. There was no change in the vision O. D. (except a little light).

Comment: This case was more chronic, but the left eye was apparently following the course of the right, which had already lost all vision. The process, which had failed to yield to all previous treatment and partial nasal surgery, subsided only after ventilation of deep-seated residual sinus pathology.

CASE 4.—A man aged 30 years. One-year history of bilateral uveitis with vision reduced to 20/200 O. U. Complete examination in several institutions was reported as negative except for a chronic pharyngitis and a postnasal drip, but the condition continued to worsen in spite of treatment. March 10, 1952, bilateral internal ethmosphenoidectomy. March 21, 1952, uveitis has subsided and vision in the right eye improved to 20/100. Several months later K. P.'s, which had previously been a trouble-

some feature, disappeared and vision O. U. improved to 20/50.

Comment: This patient was fortunate in that the uveal inflammation was arrested in time so that he retained sufficient vision to be employed again. It is noteworthy that the duration of the process and the extent of the recovery were the same in both eyes.

Case 5.—A woman aged 24 years. Fifteen months' history of uveitis in the right eye, complicated by cataract. Has daily headaches, frontal, temporal, and behind both eyes. Coughs and brings up phlegm, especially in the morning. All examinations including blood tests, x-rays, and tuberculin tests were found to be negative. The patient's present concern is the constant pain in the right eye, which is getting progressively worse in spite of treatment. Dec. 4, 1950, bilateral internal ethmoidectomy. The uveitis subsided rapidly, and six months later cataract extraction was done without incident. There has been no return of the uveitis.

Comment: One year after sinus surgery the patient developed an irritation in the left eye which followed an upper respiratory infection, which, however, yielded to conservative treatment. Five years after operation the patient showed a tendency to increased tension in both eyes, but this, too, was easily controlled by medication.

Uveitis, Postoperative or Traumatic

Chandler[12] states: "It is a well-known fact that trauma of any sort in a predisposed eye will usually precipitate an attack of iritis or uveitis. Thus the trauma of uncomplicated cataract may cause a flare-up in an eye known to have previously been affected by iritis or uveitis. However, in eyes with no history of previous iritis or uveitis a severe attack may follow cataract extraction, and if the operation is done on the fellow eye, that eye usually suffers a similar attack, thus indicating that the eye was predisposed to inflammation and only awaited sufficient trauma to precipitate the attack." The predisposing factor, referred to by Chandler, often lies in nasal pathology; at least my clinical experience seems to point that way. This is best illustrated by Cases 6 to 11 inclusive.

Case 6.—A man aged 47 years. History started 30 years ago with keratitis O. D., followed by iritis and scarring of the cornea. Three years later the O. S. became similarly affected. When first seen June 8, 1948, the patient had advanced conjunctivitis and uveitis O. U. Vision O. D. counting fingers at 3 ft., O. S. counting fingers at 1 ft. All attempts at improving the vision by surgery were unsuccessful, with recurrent flaring up of the uveal inflammation and further deterioration of the condition. Several previous attempts at clearing the sinus focus by intranasal surgery had failed. After pansinusectomy, July 18, 1950, the uveitis cleared completely, and the patient was able to have corrective ocular surgery, including successful corneal transplantation, with vision O. D. improved to 20/60.

Comment: In this case both eyes had apparently been sensitized by previous attacks of inflammation so that they could not tolerate ocular surgery. Intranasal surgery was not sufficient to eliminate the sinus pathology, probably because there was extensive pneumatization of the sinuses with extremely multilocular frontal sinuses which could be fully ventilated only by pansinusectomy.

Case 7.—A man aged 45 years. History of iritis and glaucoma of the left eye beginning three years ago. He had suffered an acute attack of glaucoma six months ago, for which he had had surgery. The patient had undergone many examinations and treatments, including cortisone, which was used systemically and locally, but for the past four months the left eye had been intensely injected, extremely painful, with marked lacrimation and photophobia. The pertinent findings after an exhaustive investigation at the most recent institution where he was hospitalized were reported as follows: Left eye—(1) localized keratitis; (2) recurrent iritis and secondary glaucoma; (3) anteriorly dislocated lens; (4) iridectomy postoperative. Nose shows evidence of vasomotor rhinitis. There is no pus present at this time, and, although x-ray examination revealed clouding and thickening of the lining membranes of both antra and ethmoids with similar involvement of the left frontal and left sphenoid, the laryngological examination at this time discloses no evidence of an active sinus infection. The roentgenological findings are probably the residuals of an acute sinusitis of several months ago. Allergy: Allergic survey was negative. There is no eosinophilia. Blood agglutination reactions for brucellosis, negative. Agglutination reactions for Melitensis group were negative. Kahn test for syphilis was negative. The only factors that were considered of possible etiological significance were (*a*) a positive Mantoux test, although there was no clinical or x-ray evidence of

organic pulmonary disease; (*b*) an extensive periodontoclasia. A mild depression was found to contribute to the production and exaggeration of the patient's symptoms. The recommendations were for control of iritis and keratitis by eradication of foci of infection and cortisone subconjunctivally. Removal of the dislocated lens to be considered later, when the ocular condition had become quiescent. However, under this regimen the uveal inflammation did not subside but instead became steadily worse until Sept. 29, 1953, when left internal ethmosphenoidectomy was done, following which the left eye became quiescent. Later left cataract extraction was performed without incident, and the patient, a watchmaker, was able to resume his occupation.

Comment: This patient had arrived at an impasse; the intense congestion of the uveal tissues precluded surgical removal of the cataract, and there could be no doubt but that the presence of the dislocated lens was continuously aggravating the inflammatory irritation already present. After the long previous history, the promptness with which the eye responded to the nasal surgery, and the uneventful recovery after the cataract extraction, would seem to establish the paranasal sinuses as the original source of the ocular inflammation.

CASE 8.—A man 58 years of age. First seen Dec. 1, 1947. Had had cataract removed from left eye June 4, 1947, after which he developed uveitis in the left eye which had persisted in spite of treatment, including fever therapy and removal of suspected teeth. Patient had also suffered from pain in the left side of face and frontal headaches. Examination revealed a thickened septum and a greatly enlarged left middle turbinate with obstruction of the upper nasal passages on the left side and polyps in the left middle meatus. Antrum irrigations showed a moderate amount of pus in the right antrum and a great deal of broken-down purulent material in the left antrum. The left eye began to improve after the first antrum irrigation. Subsequently, left internal ethmoidectomy was done, with complete subsidence of the uveitis.

Comment: Obvious maxillary sinusitis and deep-seated ethmoiditis combined to predispose this eye to inflammation when triggered by ocular surgery.

CASE 9.—A woman aged 50 years. Recurring bouts of iridocyclitis since 1947. Began in the right eye, but several years later the left eye also became involved in spite of intensive treatment and removal of infected tonsils. The bilateral uveal inflammation continued until the vision was reduced to counting fingers at 8 in. in the right eye and only light in the left eye. Besides the loss of vision the patient suffered constant pain and discomfort in both eyes, which were markedly congested. Feb. 11, 1954, bilateral internal ethmosphenoidectomy was done. Shortly after this procedure all signs of orbital infection receded, and finally both eyes whitened and the patient became asymptomatic. Later cataract extraction O. D. was done without incident and the vision in the right eye improved to 20/200.

CASE 10.—A woman aged 34 years. Bilateral uveitis. Vision O. U. had been failing for the past 20 years in spite of all treatment, including antisyphilitic therapy. Six years ago lost what little vision she had after childbirth. There was no central vision in the right eye, with perception for light only at sides. The left eye was lost, following several operations for cataract extraction and resulting complications about one year ago. May 17, 1954, bilateral internal ethmosphenoidectomy. Following the sinus surgery cataract extraction O. D. was performed without incident, and the vision O. D. improved from c. f.—1 ft. to 20/200.

Comment: While the result in Cases 9 and 10 leaves much to be desired, it is still very encouraging to know that some improvement can be obtained in advanced cases such as these after elimination of sinus pathology. It is surprising how much rehabilitation occurs even with such modest improvements in those at or near total blindness. One cannot help but be impressed by the difference in the results of cataract extraction, in the left eye before sinus surgery and that following cataract extraction in the right eye after sinus exenteration, in Case 10.

CASE 11.—A man aged 47 years. Four months' history of recurrent inflammation in the right eye. Ocular inflammation was started by a foreign body which entered the right eye while patient was driving a taxi. The foreign body was removed but the inflammation continued. The patient was treated intensively, among other things, with cortisone locally and systemically, but the condition continued to degenerate, and the ocular discomfort was beginning to spread to the left eye. Dec. 13, 1955, right internal ethmoidectomy. The inflammation in the right eye cleared promptly, and the patient was able to return to his occupation.

Comment: It may be concluded that this was a sensitized eye which was triggered into active inflammation by the trauma of

the foreign body and its removal. It may reasonably be assumed that the sensitizing agent was in the sinus pathology, which was eliminated. This patient had one brief recurrence of the symptomatology about one year later following an upper respiratory infection, but this yielded readily to conservative treatment. There has been no recurrence since.

Iritis

CASE 12.—A man aged 55 years. Had had attacks of iritis O. S. for 15 years. First seen on Aug. 10, 1951; present attack started four months ago. Treatment has been hampered by his being allergic to atropine. The patient, an actor, had been forced to give up all screen work because of intense photophobia, especially under Klieg lights and was able to do some stage work, only by using epinephrine eye drops 1-100 several times during a performance to whiten the eye. Travels a good deal and has been treated in many cities without improvement. Bilateral antrum irrigation—purulent return from both sides; vision O. S. 20/100. Aug. 17, 1951, left internal ethmoidectomy. Oct. 22, 1951, eye much quieter; O. S. 20/40. Dec. 6, 1951, O. S. 20/40, asymptomatic; Jan. 17, 1952, O. S. 20/30-1, no symptoms. There has been no recurrence of iritis, and the patient was able to return to screen work.

Comment: The long history and the rapid and permanent subsidence of the iritis precluded the possibility of coincidence of the nasal surgery and the clearing of the ocular involvement. This patient, besides the deep-seated nasal pathology had obvious bilateral purulent maxillary sinusitis, which had gone untreated and which no doubt contributed a great deal to the ocular inflammation.

CASE 13.—A woman. Affected with recurrent iritis in O. D. for past seven years; at present also has a patch of active chorioretinitis in O. D. This is the fifth attack and is of four weeks' duration. Vision in the right eye was lost after the first episode. There has been one attack of iritis in the left eye, which lasted a month but did not injure the vision. Patient was given a series of bilateral antrum irrigations for purulent maxillary sinusitis, and this was followed, Aug. 23, 1954, by right internal ethmosphenoidectomy. The intense congestion in the right eye began to subside after the second antrum wash and cleared completely after the sinus surgery. There has been no recurrence of iritis in either eye.

Comment: The situation here was made extremely grave by the fact that the uveal inflammation was threatening the only functioning eye remaining to the patient. This case is still being observed, and, should there be another attack of iritis, especially if it occurs in the left eye, a left ethmosphenoidectomy would be done.

CASE 14.—A man 55 years of age. Recurrent attacks of iritis since 1937. Started in the left eye, but has had attacks in both eyes, ranging from six months to one year apart. Had had sinus trouble for 10 years. Was operated on for this some months ago. Has been hospitalized and given all manner of treatment, including fever therapy and corticotropin (ACTH) to no avail. First seen Jan. 3, 1951. Present attack of iritis is bilateral and has lasted three months. After bilateral internal ethmosphenoidectomy, the iritis subsided, and there has been no recurrence since. The patient, completely incapacitated previously, was able to return to his occupation soon after the nasal surgery.

Comment: As a result of the long past history of uveal inflammation, irreversible changes in the left eye caused reduction of the vision to 20/200, but in the right eye the vision remained unimpaired at 20/20.

Keratitis

CASE 15.—A woman aged 39. Left eye affected by sclerokeratitis, which has been under treatment continuously for more than a year without relief. Treatment included cortisone locally and systemically. Patient is subject to frequent upper respiratory infections and has had attacks of occipital headache for years. Complains of stuffiness of the nose and that the bony structures of the nose and the face are sensitive to touch. General examinations on several occasions were found to be negative, except for a mild sinusitis, which was treated with nose drops and cortisone. The patient is very much alarmed because recently the right eye is becoming irritated. Nov. 30, 1953, left internal ethmoidectomy was done, after which all the symptoms subsided, and there has been no recurrence since.

Comment: Here again we have the combination of unabated distressing ocular inflammation with unmistakable nasal symptoms, which were of sufficient mildness to be controlled symptomatically by palliative treatment, without, however, improving the ocular condition. The latter cleared quickly, once the residual sinus infection was eliminated.

CASE 16.—A woman aged 48. Has had keratitis in the left eye for the past three months with intense photophobia. Had had an attack of iritis in the right eye, which subsided just before the

onset of the present difficulty in the left eye. The previous history was otherwise negative, except for occasional head colds and frontal and occiptal headaches and an attack of pleurisy with effusion 12 years ago. On the basis of the pleurisy, the eye condition was diagnosed and treated for tuberculosis, although repeated chest x-rays were negative. Jan. 24, 1950, bilateral internal ethmoidectomy resulted in only partial relief of the ocular symptoms, but even this proved temporary. Three months later right pansinusectomy was done, with complete subsidence of the ocular inflammation.

Comment: This case illustrates that the focus of the infection may be contralateral to the eye involvement and so deeply seated that it was not uncovered by the first operation, which was intranasal.

Case 17.—A woman aged 22. First seen Aug. 24, 1949. Keratitis O. D. Here again, right internal ethmoidectomy Aug. 31, 1949, failed to clear the condition, and the patient suffered with recurrence of the keratitis, which, however, was milder than previously. Sept. 13, 1952, right external frontoethmosphenoidectomy was done for the exenteration of residual sinus pathology. There has been no further recurrence of the keratitis to date.

Comment: Coincidence of the nasal surgery and subsidence of the ocular inflammation can logically be ruled out here as there was a lapse of three years between the internal and external operation with a further lapse of five years since the last attack of keratitis.

Case 18.—A woman aged 48. Recurrent keratitis. Lacrimation, orbital pain, photophobia, and congestion in the left eye for several months and also herpes of the left side of the nose. Poor vision in left eye since childhood, with opacity over the pupil, which had been getting larger the past 15 days. Examination O. S.; intensely congested, corneal opacity covering pupillary area. Feb. 25, 1952, left internal ethmosphenoidectomy. The herpes disappeared shortly after the operation. The keratitis cleared gradually; by March 21, 1952, the left eye was completely white. There has been no recurrence of the keratitis or herpes to date.

Comment: Evidence of trigeminal nerve involvement is furnished by this case, where the keratitis was associated with nasal herpes and both responded to correction of nasal pathology.

Case 19.—A boy aged 15. Eight months' history of congestion, burning, lacrimation, discharge, and extreme photophobia O. U. due to keratitis. Had been hospitalized and given all sorts of treatment including corticotropin and cortisone (both intramuscular and in eye drops.) also beta radiation without improvement. Patient had had no treatment since Nov. 23, 1950, because his mother was discouraged with the lack of progress. To minimize his ocular discomfort he had been kept at home in a completely darkened room. Examination O. U. showed edema of lids, marked congestion of tarsal conjunctiva; O. S. more advanced with gelatinous appearance and congestion of the bulbar conjunctiva. Vision could not be determined; it was impossible to get patient to open his eyes. The nose was completely blocked and showed evidence of advanced sinusitis. Bilateral antrum irrigation gave copious purulent return from both sides. Jan. 10, 1951, bilateral internal ethmoidectomy and sphenoidectomy. Almost immediately after operation there was a marked improvement in all his symptoms, and the patient was able to tolerate all but extremely bright light. Jan. 24, 1951, no photophobia or lacrimation, O. U. quiet, O. D. 20/200, O. S. 20/40.

Comment: Distressing ophthalmic inflammation is no respecter of youth, but, when it strikes, obvious nasal pathology will more often be found than is the case with adults. In this patient irreversible changes in the ocular tissues had already taken their toll before the nasal pathology was corrected.

Case 20.—A woman aged 43 years. Recurrent attacks of keratitis in the left eye for 14 years. The left eye has developed glaucoma which necessitated many operations for tension. Tonsillectomy had been done four years ago, and submucous resection one year later without relief. The patient had also had several series of tuberculin injections, although there was no evidence of tuberculosis. Vision in the right eye has been poor since childhood. Feb. 2, 1948, left external ethmoidectomy was followed by prompt subsidence of the ocular congestion and glaucoma. There has been no recurrence of tension in the left eye, and the patient, a nurse, was able to return to her occupation.

Comment: In this case the sinusitis was obvious with copious purulent discharge in the left middle meatus. The submucous resection previously done had failed to clear the sinusitis because, as was evident at operation, adhesions and obstruction persisted deep in the left upper meatus. Shortly after complete exenteration of the ethmoid cells, the nasal discharge ceased. There has been no recurrence of the glaucoma, which in common with the keratitis apparently had its etiology in sinus pathology.

Macula Edema

CASE 21.—Patient aged 37 years. The patient had had two attacks of macula edema in the right eye in the past year and a half. Gives a history of sinusitis, which was treated, and had had middle turbinectomy and submucous resection some years ago. Had been treated with cortisone but the macula edema is increasing. June 16, 1954, right internal ethmosphenoidectomy. Two days after the sinus surgery the macula edema began to recede, and there has been no recurrence since. Vision O. D. has remained at 20/20.

Comment: The posterior sinuses are at times in intimate relation with the retina. Here apparently ventilation of an obstructed posterior ethmoid cell caused subsidence of the macula edema.

Summary

The frequency with which nonspecific endogenous ocular inflammation responds to correction of nasal pathology could indicate a causal relation between the two. The difficulties encountered in diagnosing deep-seated sinus infection, which is often asymptomatic, are detailed. Possible routes of transmission of infection from the sinuses to the eyes are outlined, but actual proof, other than clinical, of such transmission is not possible at the present stage of our knowledge.

This approach to the problem of nonspecific endogenous ocular inflammation was used in each case only after all known methods of treatment, including elimination of other foci of infection, had failed to halt the progress of the disease. There has been no recurrence of the ophthalmic inflammation in any of the patients, although the follow-up averages more than five years.

Ophthalmologists refer to a predisposing factor as explaining uveal inflammation following cataract extraction and other ocular surgery. A prior attack of iritis or uveitis is said to account for some of these sensitized eyes, but the cause in many of the others, with no previous history of uveal inflammation, may be in concomitant nasal pathology. Case reports were utilized to illustrate the various phases of the subject.

Conclusion

My clinical experience would seem to indicate that nasal pathology, obvious or latent, is the predisposing or sensitizing factor in many cases of nonspecific endogenous ocular inflammation, spontaneous or that triggered by trauma, operative or accidental. In the face of ophthalmic inflammation with progressive loss of vision, where no cause can be found and no treatment is effective, ventilation of the nasal sinuses is strongly advocated, even if only as a diagnostic procedure. Where both eyes are involved or the affected eye is the only one remaining, such intervention becomes all the more urgent and should not be too long delayed, lest irreversible changes occur in the ocular tissues, with permanent loss of sight.

960 Park Ave. (28).

REFERENCES

1. Lancaster, W. B., and Foote, F. M.: The Battle Against Blindness, J. A. M. A. 145:26, 1951.

2. Zamorani, G.: Clinical and Experimental Contribution to Knowledge of the Nasoretinal Reflex, Riv. oto-neuro-oftal. 24:423, 1949.

3. Schaeffer, J. P.: The Nasal and Paranasal Relations of the Membraneous Lacrimal Passageways, in The Nose, Paranasal Sinuses, Nasolacrimal Passageways, and Olfactory Organ in Man, Philadelphia, P. Blakiston's Son & Co., 1920, p. 253.

4. Schaeffer, J. P.: The Sinus Sphenoidales, in The Nose, Paranasal Sinuses, Nasolacrimal Passageways, and Olfactory Organ in Man, Philadelphia, P. Blakiston's Son & Co., 1920, p. 193.

5. Johnson, H. P.: Diagnosis of Chronic Maxillary Sinusitis, Arch. Otolaryng. 5:309, 1927.

6. Semenov, H.: Pathology of the Nose and Paranasal Sinuses, Tr. Am. Acad. Ophth. 57:399, 1953.

7. Kern, R. A.: Discussion of Etiology and Nature of Chronic Hyperplastic Sinusitis, J. Allergy 4:545, 1933.

8. Hansen-Pruss, O. C.: Bacterial Allergy and the Importance of Otolaryngological Procedures, Laryngoscope 59:540, 1949.

9. Castroviejo, R.: Ocular Allergy Tr. Am. Acad. Ophth. 56:242, 1952.

10. Rosenow, M. J.: Focal Infection and Elective Localizations in Pathology of Disease of the Eye, Tr. Am. Acad. Ophth. 32:4, 1927.

11. Woods, A. C.: Use of Specific Streptococcus Vaccine in Nongranulomatus Uveitis, A. M. A. Arch. Ophth. 50:129, 1953.

12. Chandler, P. A.: Cataract Extraction, Tr. Am. Acad. Ophth. 58:382, 1954.

Alcoholic Beverage, Nasal Temperature, and the Common Cold

NOAH D. FABRICANT, M.D., Chicago

Alcoholic beverages have been utilized for generations to abort the common cold, especially after chilling by exposure to inclement weather. Fear of increasing their popularity has prevented many physicians from prescribing alcoholic beverages during the prodromal stages of the common cold. Without contending that such a practice is effective or that it can stay the self-limited course of the common cold, Goodman and Gilman [1] point out that alcohol exerts several actions which may prove beneficial, even though they are not essential. Alcohol causes peripheral vasodilatation and reestablishes circulation in chilled cutaneous and mucosal surfaces; provides comfort; induces drowsiness, and promotes a desire to rest. Once acted upon, the decision to rest in bed can serve a most useful purpose. Rest in bed diminishes the severity of the common cold, limits its spread to others, and reduces the frequency of complications.[2]

It has been demonstrated by Mudd and his associates [3] that chilling the body surface causes three phenomena: a fall in the topical temperature of the nasal mucosa, a pronounced reflex vasoconstriction, and a diminution of the blood supply. According to Spiesman,[4] a fall in the topical temperature of the mucosa of the upper respiratory tract indicates vasoconstriction, while a rise in temperature indicates vasodilatation. Chilling the body surface, moreover, produces ischemia of the mucosa. When subjected to the influence of an infectious environment, an ischemic nasal mucosa can become infected as a direct consequence of the lowered resistance produced by prolonged ischemia and anoxemia. Indeed, abnormal vasomotor disturbances of the mucosa not only cause local injury to the tissue but also predispose to infection by pathogenic agents already present.

A fall in the topical temperature of the nasal mucosa, vasoconstriction, and ischemia occur during the prodromal stages of the common cold. Vascular spasm, ischemia, and anoxemia injure the nasal defenses and pave the way for acute infection. Clinically, this is translated into a sensation of nasopharyngeal dryness and focalized discomfort, diminished nasal secretions and impaired mucociliary activity. A prime objective of therapy during the earliest stages of the common cold is to restore the nasal mucosa to a normal, physiological state. Agents that raise the topical temperature of the nasal mucosa, produce vasodilatation, and combat localized mucosal chilling are desirable.

Present Investigation

In the present investigation, the topical temperature of the nasal mucosa was determined in 10 clinically normal adults (6 men and 4 women) and in 2 adults (1 man and 1 woman) in the prodromal stages of the common cold, before and after they had consumed a measured amount of an alcoholic beverage. Topical temperature measurements of the right and left sides of the nasal cavity were taken by the Rauh Surface Pyrometer, a device for making rapid surveys of the mucous membrane and cutaneous temperatures of the human body. The topical temperature of the nasal mucosa was obtained by slight contact of the sensing element with the anterior one-third of the inferior turbinate. Various precautions were observed to assure accurate readings; these have been described in a previous communication.[5] Nasal temperature readings were taken immediately before consumption of the alcoholic beverage and repeated after consumption of the alcoholic beverage at intervals of 15 minutes for a period of one hour.

Submitted for publication Oct. 3, 1957.

Data on the Effect of an Alcoholic Beverage on the Nasal Temperature of Twelve Adults, Before and After Consumption

Patient	Sex	Before Consumption		After 15 Minutes	After 30 Minutes	After 45 Minutes	After 1 Hour
1	F	Right nasal passage	89.0 F	90.5 F	91.0 F	92.0 F	93.0 F
		Left nasal passage	90.0 F	90.5 F	90.5 F	91.0 F	92.0 F
2	M	Right nasal passage	89.5 F	89.5 F	92.0 F	92.0 F	92.5 F
		Left nasal passage	91.0 F	90.5 F	93.5 F	92.5 F	92.0 F
3	M	Right nasal passage	88.0 F	88.5 F	89.0 F	89.0 F	90.0 F
		Left nasal passage	90.0 F	90.5 F	90.0 F	91.0 F	92.5 F
4 *	F	Right nasal passage	85.0 F	89.5 F	89.5 F	92.0 F	92.0 F
		Left nasal passage	83.5 F	87.0 F	88.0 F	92.0 F	92.5 F
5	M	Right nasal passage	90.0 F	92.0 F	92.0 F	91.5 F	93.5 F
		Left nasal passage	90.5 F	93.0 F	93.5 F	92.0 F	93.0 F
6	F	Right nasal passage	90.0 F	91.5 F	92.5 F	92.0 F	93.0 F
		Left nasal passage	89.0 F	91.0 F	91.0 F	93.0 F	92.5 F
7 *	M	Right nasal passage	86.0 F	87.5 F	88.0 F	88.5 F	90.0 F
		Left nasal passage	84.0 F	88.0 F	89.0 F	89.0 F	91.0 F
8	F	Right nasal passage	91.0 F	93.0 F	93.0 F	93.0 F	93.5 F
		Left nasal passage	91.0 F	94.0 F	94.5 F	94.0 F	94.5 F
9	M	Right nasal passage	89.0 F	89.5 F	91.0 F	92.0 F	92.0 F
		Left nasal passage	90.5 F	90.5 F	92.0 F	91.5 F	92.5 F
10	M	Right nasal passage	90.0 F	90.5 F	91.5 F	92.0 F	93.5 F
		Left nasal passage	91.5 F	92.0 F	92.5 F	93.5 F	93.0 F
11	F	Right nasal passage	88.5 F	88.5 F	91.5 F	90.0 F	92.0 F
		Left nasal passage	89.0 F	90.5 F	91.0 F	90.5 F	91.5 F
12	M	Right nasal passage	89.5 F	90.5 F	92. 0F	93.0 F	92.0 F
		Left nasal passage	90.0 F	90.5 F	92.5 F	92.0 F	92.5 F

* Patients 4 and 7 were in the prodromal stages of the common cold.

The alcoholic beverage used in this investigation was standardized to contain 1 oz. of a blended whisky. Blended whisky contains a smaller percentage of congeners (the ingredients that give alcoholic beverages their taste, aroma, and color) than do other types of whisky. It has been determined that too high a concentration of congeners can produce certain after-effects such as "hangover." The present investigation was conducted at a room temperature that varied slightly, from 70 to 72 F (Table).

Comment and Summary

Alcoholic beverages have been utilized for generations to abort the common cold, especially after chilling by exposure to inclement weather. Fear of increasing their popularity has prevented many physicians from prescribing alcoholic beverages during the prodromal stages of the common cold. Alcohol exerts several actions that may prove beneficial. Alcohol causes peripheral vasodilatation and reestablishes circulation in chilled cutaneous and mucosal surfaces; provides comfort; induces drowsiness; and promotes a desire to rest.

Chilling the body surface produces a fall in the topical temperature of the nasal mucosa, a pronounced reflex vasoconstriction, ischemia, and a diminution of the blood supply. These phenomena occur during the prodromal stages of the common cold. Vascular spasm, ischemia, and anoxemia injure the nasal defenses and pave the way for acute infection. A prime objective of therapy during the earliest stages of the common cold is to restore the nasal mucosa to a normal, physiological state. Agents that raise the topical temperature of the nasal mucosa, produce vasodilatation, and combat localized mucosal chilling are desirable.

In the present investigation, consumption of an alcoholic beverage standardized to contain 1 oz. of a blended whisky was found to produce a rise in the topical temperature of the nasal mucosa of 12 adults (7 men and 5 women), 2 of whom were in the prodromal stages of the common cold. The ability to produce a rise in nasal temperature indicates the physiological usefulness of an alcoholic beverage during the prodromal stages of the common cold. Although consumption of alcohol is obviously not a cure for the common cold, its beneficial role in some persons can neither be minimized nor dismissed.

185 N. Wabash Ave. (1).

REFERENCES

1. Goodman, L. S., and Gilman, A.: The Pharmacological Basis of Therapeutics, Ed. 2, New York, The Macmillan Company, 1955.

2. Fabricant, N. D.: Patient's Plight, Doctor's Despair—The Common Cold, Am. J. Med. Sc. 214:458, 1947.

3. Mudd, S.; Goldman, A., and Grant, S. B.: Reactions of the Nasal Cavity and Postnasal Space to Chilling of the Body Surface: I. Vasomotor Reactions, J. Exper. Med. 34:11, 1921.

4. Spiesman, I. G.: An Experimental and Clinical Study of the Common Cold, Ann. Otol. Rhin. & Laryng. 50:1204, 1941.

5. Fabricant, N. D.: The Topical Temperature of Clinically Normal Nasal and Pharyngeal Mucous Membranes, A. M. A. Arch. Otolaryng. 66:275, 1957.

Psychogenic Vertigo

The Importance of Its Recognition

BURNESS E. MOORE, M.D., and MILES ATKINSON, M.D., New York

Severe vertigo is a vastly distressing symptom, and understandably may give rise to acute anxiety. But such anxiety is secondary to the disease and is based on the reality of disturbed equilibrium. This paper deals with a different sort of anxiety, anxiety which precedes and is the cause of the symptom of vertigo instead of being its result. It is based on neurotic conflicts which are unrecognized by the patient. For the physician to be aware that such a condition exists and to be able to recognize it is of great importance, for not only can it be seriously, even totally, disabling, but it will obviously not respond to the usual medical or surgical procedures. In fact, prolonged and unsuccessful otological treatment may even result in fixation of the neurotic symptom.

Though fairly common, it seems to us that vertigo of purely psychic origin has not received sufficient attention from either otologists or psychiatrists. It is the purpose of this paper, by an otologist and a psychiatrist, to bring to notice certain features of the condition which will help to clarify the respective roles of both specialties in their joint responsibility for diagnosis and treatment.

The 34 patients in the series upon which this report is based were referred initially to the otologist with a diagnosis of organic disease. After thorough clinical investigation had suggested a psychological basis for the symptoms, each patient was referred to the psychiatrist for further study. In the first part of this paper the means will be presented by which we believe vertigo of primarily psychogenic nature may be recognized by the otologist. Without such recognition and referral the psychiatrist will see relatively few cases. In the second part of the paper an attempt is made to give a simplified psychopathological explanation of the symptoms as understood by the psychiatrist. A few brief clinical vignettes illustrate how such psychological concepts may be used in understanding the patient's problems and the meanings of the symptoms.

Submitted for publication Aug. 3, 1957.

Read before the Sixth International Congress of Otolaryngology, Washington, D. C., May 6, 1957.

Associate Clinical Professor of Psychiatry, Yale University School of Medicine (Dr. Moore); Associate Professor of Clinical Otolaryngology, New York University Post-Graduate Medical School (Dr. Atkinson).

Differential Diagnosis

It is usually not difficult to distinguish the psychogenic from the organic case, given, as we have said, awareness of the possibility. Indeed, we would say with Shakespeare that the readiness is all. To be more specific, three main points distinguish the psychogenic from the organic case:

1. The type of vertigo
2. An absence of physical signs
3. Indications of emotional disturbance

The Type of Vertigo.—We would stress the importance of a scrupulously careful history. Simple acceptance of the phrase "dizzy attacks" is not enough. The type of sensation must be more explicitly determined. It is traditional to state that vertigo is organic when it is external rotatory (the world spins); that it is psychogenic when it is internal rotary (the patient spins). While suggestive, we believe such distinctions tend to be used as shibboleths. They should not be regarded as the ultimate criterion.

Whether organic or psychogenic, vertigo may be related to position and be accompanied by a sensation of imbalance or insecurity. In this connection, it must be remembered that patients like to give histories in scientific terms, but that they often distort the meanings of these terms to fit their own ideas. What the patient calls vertigo may turn out to be faintness, unsteadiness, light-headedness, "floating in space," or a momentary flash. In one case, vomiting, a common enough word with well-accepted implications, was revealed to mean "saliva running out." Such distortions must make one beware of taking the patients' words at face value.

The neurotic patient, especially, will often seek to dramatize his symptoms, giving them respectability in the guise of scientific jargon. A more detailed description of the symptoms in the patient's own language will sometimes provide important data, totally inconsistent with organic syndromes, but highly relevant to the hidden psychological meaning of the illness.

Absence of Physical Findings.—A thorough, painstaking physical examination must be made, for even an obvious neurotic may have organic disease. As someone very cogently said, "Neurotics also die." It is as serious a mistake to treat organic disturbance psychiatrically as to treat a psychological disturbance organically. In each case the result may be disastrous. But in the absence of physical findings in the four systems mainly responsible for vertigo—the vestibulocochlear, the nervous, the cardiovascular, and the hematological—one should at once suspect that the condition is psychogenic and proceed to the search for proof.

Indications of Emotional Disturbance.—In the field of psychogenic disorders, as in the field of organic disease, a diagnosis must be made from positive findings, not from their absence and an inability otherwise to explain symptoms.

Positive evidence of psychogenicity may be very easy to obtain or very difficult. The primary requisite is that the patient be given a chance to talk. Given the opportunity, perhaps the most characteristic feature of the patient with psychogenic vertigo is the way in which he tells his story. Very often he is loquacious and verbose, giving lengthy and bizarre descriptions of his sensations, such as the following: "Suddenly I seem to be spinning off into space." "The top of my head feels ready to spin off." "The dizziness races from my head to the pit of my stomach." "There is a tingling up and down my spine." These dramatic descriptions may be given with a cheerful smile, recalling "la belle indifference" of the hysteric, or they may be accompanied by an unmistakable show of emotion in the form of tears, blushing, or general agitation. Interspersed with the description of symptoms may be accounts of family conflicts, disturbing early experiences, and questions of moral conduct, seemingly irrelevant to the problem, but obviously connected with the symptom in the patient's mind.

On the other hand, some patients are uncommunicative and even hostile to the point that nothing more than the simplest statement of dizziness can be obtained, at least at the beginning. Such reserve, or a vehement, angry denial of any connection between physical symptoms and emotional feelings, should at once arouse suspicion of a psychological basis for the complaints. "The lady doth protest too much, methinks." In still other cases, such flagrant indications of emotional disturbance are absent, and one encounters an impassive, phlegmatic person, devoid of imagination and introspection, concerned apparently only with practical, concrete, everyday problems. It may take long contact and the most discreet probing before the significant information is disclosed, sometimes, it would seem, almost inadvertently. Since the patient cannot cooperate by facing the problem, diagnosis of the emotional factors may have to remain uncertain. For these difficult cases, one simply has to develop a nose.

The circumstances of the first attack, or special features which are recurrent, may immediately point to a neurotic origin. If the attacks occur at the same time of the day or night (though in this connection, beware the hypoglycemic attack), in a particular place, or under particular circumstances, this story may be highly significant. We recall, for instance, the woman whose attacks occurred only in bed, and only when she turned onto her right side. They were accompanied by nausea and even vomiting and were so severe that she would have to leave the bed which she shared with her husband, whose place was on her right. There was much marital discord. After failure of conventional therapy, twin beds greatly diminished the frequency and severity of her attacks, and finally divorce cured them. Another patient was free of all symptoms when on vacations, only to have a prompt recurrence when she was joined by her husband.

The Basis for Psychological Symptoms

A brief description of the basis of psychological symptom formation will be of help in understanding the nature of psychogenic vertigo. From physiology we know that many factors, such as stress or infection, may disturb the normal functioning of organs or systems, and the body has a number of automatic controls and defenses with which to restore the former balance. This homeostatic concept is equally applicable to the functioning of the human mind. In this case, however, instead of infection or oxygen imbalance, the disturbing influences consist of impulses stimulated by sensory perceptions, memories, or feelings. Many of the impulses are sexual or aggressive in nature and totally unacceptable to the individual or society; their existence therefore gives rise to anxiety, which is a signal of danger, calling forth psychological defense mechanisms to deal with them. A conflict arises between the primitive instinctual drives struggling for expression and what might be called the forces of conscience opposing them. When these forces are equally matched, neither can win, and a homeostatic solution of the conflict is reached by a compromise between the forbidden wishes and the forces of conscience, similar to the pathology which is the end-result of the struggle between disease and the defenses of the body. Though the patient is no more aware of mental struggle and the resulting psychopathology than he is of specific organ pathology, these internal processes are manifested by symptoms which may be experienced by the patient as physical or mental. In psychological terms a symptom may represent the gratification of a forbidden wish in a distorted symbolic way, or the defenses against the wish, or punishment for it.

The several different types of neuroses are named in accordance with the nature and function of the principal symptom and the psychological defenses employed, for example, hysteria and phobias. However, these types are seldom pure. For clarification a brief and somewhat oversimplified explanation of the basis for such hysterical and phobic symptoms will be given with examples, since these neurotic problems are most commonly associated with psychogenic vertigo.

In hysteria mental ideas and feelings about sexual matters are converted into physical feelings in a nonsexual organ, in the present context, the ear or head. A male patient in psychoanalysis realized one day the nature of the defenses he had built up to avoid conscious awareness of his castration fears. These fears had arisen in relation to specific threats in his childhood but had persisted in all their strength because during childhood they were so frightening he had banished them from consciousness, and until now he had been unable to dispel them by conscious reexamination in the light of adult knowledge. Immediately after becoming conscious of his fears he remarked on the fact that he visualized a small boy naked. For an hour or so after the session he experienced scotomata which made him

virtually blind. The next day he confessed that he did not tell me the preceding day that the naked little boy he visualized had turned into a little girl, i. e., without a penis. He recalled that during his childhood he had often had fears of injury to his eyes. In this typical hysterical attack the blindness represented symbolically the feared loss of his genitals because of incestuous wishes. When the fears came close to consciousness, he dealt with them according to his childhood defense of displacing such fears on to his eyes, probably selected because the eyes provide an important stimulation for sexual excitement. In this case the blindness represented a punishment for the wish to see sexual things which were forbidden. On another occasion this same patient experienced an attack of dizziness when other defenses were interpreted.

In phobias a situation may be selected which is symbolically representative of the forbidden sexual wish. Thereafter exposure to such a situation produces intolerable anxiety. By avoiding the situation the patient is able to prevent anxiety about his forbidden wish and even awareness of its true nature. For example, an attractive young woman, whose unconscious wish was to be like the call girls her father was known to prefer to her mother, developed a phobia of traveling, a situation which had in the past provided her with many opportunities for gratifying her wishes. This change occurred when circumstances arose which made gratification of these wishes no longer acceptable to her. She had to avoid further gratification and even awareness of the wishes, and was able to do so by means of her phobia.

Not all neurotics have clearly definable symptoms, but it may be apparent that in their attitudes and behavior toward others they are giving expression to their impulses and defenses in an indirect way. These people, who suffer from what is called character neuroses, go through life as difficult persons who antagonize others, dominate their families, compulsively repeat their mistakes in diverse fashion, fail when they have reached the point of success, or make a career of suffering. For them their neurotic symptom consists of a way of life. If this way of life is upset, they too may develop specific neurotic symptoms. In any neurotic there is usually an admixture of various types of symptoms, one replacing another in accordance with the need and situation.

The Vertigo Cases

The cases of psychogenic vertigo we have studied showed striking diversity in their psychological problems, understandable only on the basis of the mobility of psychic processes. Although all had serious neurotic problems antedating their vertigo, they had achieved a relatively good state of psychic equilibrium until some dramatic incident or change in their general life situation disturbed their balance. As in the hysterical example given previously, dizziness as a transient symptom is frequently observed in psychiatric treatment when such a change has been produced by the interpretation of old defenses by which the patient had achieved a neurotic peace of mind. For many patients, therefore, the symptom of vertigo is a concrete symbolic representation of their upset mental state and conforms to the hysterical pattern of conversion of a mental idea into a physical feeling. This may account for the fact that most neurotics experience the phenomenon of vertigo as one of person bodily disturbance (the patient spins) rather than of the external world. However, those who attribute their faults to others may also project their own inner turmoil. Such patients described their vertigo as something violent being done to them, as in the following accounts: "I felt as though I was being thrown down." "It seemed as if the floor was coming up to hit me."

An attitude toward a person or situation may be reflected in the symptom, e. g., "You make me sick," as in the instance of the

two women who became dizzy only when in close contact with their husbands.

A 35-year-old woman, who complained of excessive fatigue in addition to dizziness, is married to a passive man, chronically ill with diabetes. They refrained from having children allegedly because of his illness, but the husband is frequently impotent and the wife is frigid. She is "stuck" in a job which she dislikes intensely but which pays her nearly twice her husband's salary. The time of occurrence of her attacks of vertigo has been particularly striking. Her first and most recent attacks were experienced while she was cleaning house on a Saturday morning, a chore she despises and her husband enjoys. Once "I looked up at my husband and felt dizzy." On another occasion she had a recurrence of dizziness when her husband was enjoying a vacation alone in Florida. The patient has not been able to go away on vacations because she has asthma whenever she does. When she developed vertigo a second time when her husband was away, he felt impelled to give up his vacations.

For this patient the vertigo, through the hysterical conversion of an attitude into a symptom, provided an aggressive expression of her resentment against her husband and her lot in life. At the same time she was punished by her suffering for having such unacceptable feelings.

Some of the patients exhibited a marked degree of hostility toward all physicians. These were principally masculine women who used their suffering as a means of aggression against the doctor and their families in revenge for their status as women. In these persons, the symptom of dizziness occurred when their domination of the family was threatened by some new development, and the symptom was used as a means of regaining control.

In some instances illness had been dramatically precipitated by a highly charged emotional situation. Two cases, which will be presented in some detail, illustrate, however, that there are often multiple emotional determinants of the illness, and that in fact life is not so simple as we would sometimes like to believe.

A 59-year-old man "keeled over" with a dizzy spell followed by nausea, vomiting, tinnitus, and deafness. At the time he was making a telephone call about the death of a close friend who was partially deaf and wore a hearing aid. Symptoms had persisted for over three years and he had remained at home for four months before he came for treatment.

His father was an alcoholic. The patient, the oldest of four children, was placed in an orphanage for two years at the time of his youngest sister's birth when he was 4 years of age. This fact was recalled by the patient and related to his wife for the first time after the beginning of psychiatric treatment. There were no strong ties between the children. The patient now has no communication with his brother, is on bad terms with his younger sister, and another sister is dead. His first marriage was to a very domineering woman, a borderline psychotic, from whom he separated seven years after their marriage. She was a revengeful person who pursued him, and he was always afraid of her. Although Catholic, he married a second time a few years after their divorce. He was worried about being outside the Church, yet he has been unable to accept his second wife's offer to remarry him in the Catholic Church after the death of the first wife. After his divorce he drank heavily for 10 years, but stopped abruptly several years ago. Since then he has been finicky about food and has smoked excessively. Though successful in business and prominent in sports (he was an Olympic competitor), the patient had always been nervous, tense, and excitable.

For a month or so before his initial attack the patient was involved in frequent arguments with his sister over the care of his mother. The mother, who was senile, had been living with his sister, and the sister wanted to commit her to an institution. The patient opposed the idea and was in a great deal of emotional turmoil. On the very day his symptoms began the patient had a long, intimate talk with the friend who died about their personal problems, particularly in regard to mothers and mothers-in-law. On the same day a niece of his wife died. A few weeks after these deaths and the onset of his vertigo the patient was notified that his mother had been committed to a mental hospital three weeks earlier. He was very much depressed by the news and suffered marked disturbance of sleep and appetite. He attributed his dizzy spells to food and gradually eliminated one food after another from his diet. He has never been able to visit his mother in the institution because he is afraid that he would "get a set-back and have more dizzy spells."

Shortly before the onset of his symptoms the patient changed his business association from a small firm to another with larger facilities. He was in considerable conflict about this because the owner of the business he left was a good friend who had been very generous to him. The change was not fortunate. The new firm went bankrupt within a year, and the patient then became associated with another business which was an inconvenient

distance from his home, nonprogressive, and slipshod about production schedules. In addition the plant superintendent was a very dependent person who brought all his problems to the patient. While going through these changes the patient's attacks of vertigo became progressively worse, until he had to give up work and was afraid to venture across the room. Always dependent upon his wife, he now would go nowhere without her.

While it is apparent that he had life-long neurotic problems, the acute onset of the symptoms of vertigo, deafness, and tinnitus occurred in a situation of severe emotional disturbance over the commitment of his mother and the death of his friend. The commitment of his mother must have represented for him a repetition of loss of her when he himslf "was committed to an institution" at 4 years of age. The same sister was responsible for the loss on both occasions. Such a separation always engenders hostility in the child, and he would experience her commitment as a sort of revenge which would inevitably produce strong guilt feelings and depression. For this reason he had to oppose the commitment even though it was clearly indicated. Death of a loved one or close friend often results in an identification with the dead person, accomplished in this case through the hysterical symptom of deafness. The dizziness reflected his unstable status when he was suddenly faced with the loss of both mother and his close friend (father) on whom he was dependent for business. Thereafter the symptoms provided him with important secondary gains in escape from an intolerable business association and in care and attention from his wife. Moreover, his suffering was felt as a punishment by which he could expiate his strong feelings of guilt.

In this case and in a few others even the deafness which was associated with psychogenic vertigo had the transparent implication of "there are things that are too disturbing to hear, and so I won't listen." These disturbing things sometimes had a sexual meaning.

A 61-year-old woman presented a history of Ménière attacks, but without significant organic physical findings. Attacks occurred in relation to emotional disturbances over the divorces of two of her children, the miscarriage of another daughter, and the sudden death of a friend. The patient and her husband are both devout Catholics, which complicated these problems for her. She had been subject to anxiety since childhood, often so vivid as to be nearly hallucinatory in nature. There were phobias and occasional periods of depression. In psychotherapy it was evident that she was suffering from a tremendous amount of guilt feeling. Finally she brought in a dream in which she had been trying to arrange her garbage so that it would not be noticeable to the neighbors, among whom she listed a doctor. The interpretation was given that there were certain thoughts and feelings of which she was ashamed and that she did not want to have come out in treatment. On her next visit she told about an extramarital affair which she had been having most of her married life with her husband's employer, and which was still continuing. A few months later she had another attack of vertigo, preceded a few days earlier by a severe attack of dizziness which her lover had while he was with her. Only at this time was the history obtained that her lover had suffered dizzy spells five years earlier, just before the onset of the patient's symptoms.

This neurotic woman evidently felt that her lover's dizzy spells were punishment for their sexual misbehavior. Her own feelings of guilt led her to identify with him in his illness and so to share his punishment with him. Thereafter the neurotic symptom became generalized and was elicited by a variety of charged emotional situations.

The high incidence of phobias in patients with psychogenic vertigo was surprising, although the association has been noted previously in the literature. In such patients the hysterical symptom of vertigo appeared as an additional solution to the conflict when situations arose which suddenly increased the strength of sexual or aggressive impulses or weakened the old controls. One patient, whose phobias protected him from his "Peeping Tom" tendencies, experienced the sudden onset of dizziness while on the way to a burlesque show. The vertigo provided further protection against the gratification of his forbidden wishes and at the same time punished him for having them. A woman with strong exhibitionistic tendencies which phobic symptoms had not entirely controlled began to have vertigo just before she was to be elected president of a large organization, a fulfillment of her exhibitionistic wishes that was marred and no longer acceptable because of an injury she had received shortly before. Fearing failure in the new position she was glad to have an illness which afforded her a face-saving escape and now found a new satisfaction in her symptoms. A young woman, warding

off her sexual impulses by means of frigidity and phobias, resorted to vertigo just after she and her husband had decided that they should have a baby. She confessed that she had been terribly frightened by stories of painful childbirth. Her own parents had been divorced just before she was born, and childbirth therefore threatened her with the further danger of losing her husband.

Therapeutic Approach

It is very evident, then, that the psychological background of such vertigo is far removed from the domain of the otologist, but the patient is unaware of this fact. Unless its true nature is recognized, medical care will not help. Such cases respond best to a combined medical and psychological approach. Though it may be the presenting symptom and the most disabling, vertigo of psychogenic nature is only one aspect of the problem of such patients. The symptom itself may have multiple psychic determinants, all unconscious, which need to be removed by long, patient psychotherapy for complete relief. This can best be accomplished in the hands of an experienced psychiatrist, trained to deal with neurotic and psychosomatic conditions. However, psychiatric treatment is not always available, and a large number of our patients were unable to accept psychiatric treatment. To some it was too great a threat; others simply had no awareness of emotional processes, and psychological treatment would have been like trying to elicit the principles of higher mathematics from a bricklayer. Recognition of such limitations must be part of the joint consideration of diagnosis and therapy by the otologist and psychiatrist. Thus treatment will in most cases have to remain in the hands of the referring physician. He need have no fears about his psychotherapy, provided that he does not attempt psychological interpretations. With an interested listener, patients proceed at their own pace and many achieve by themselves a surprising degree of understanding of their problems. The role of the physician is in itself psychologically therapeutic. If in addition he adopts also that of guide, philosopher, and friend and encourages the patient to air his problems, he will, we find, in most cases obtain a very satisfactory therapeutic response.

36 E. 36th St. (16).

Audiology Section

JOSEPH SATALOFF, ASSOCIATE EDITOR

Premedication in Clinical Audiometry

An Investigation of the Effect of Mephenesin Carbamate (Tolseram) on Normal Hearing Thresholds as Determined by the Conditioned Psychogalvanic Skin Response and Conventional Pure-Tone Audiometry

RAYMOND B. STRAUSS, Ph.D., Cleveland

Introduction

With the development of the pure-tone audiometer has come an improved and increasingly more reliable hearing test technique for evaluating the auditory acuity of the hard of hearing adult. At the same time, the inadequateness and unreliability of this means of testing the hearing of infants, children, malingerers, and persons with psychogenic deafness has been acknowledged.[1]

The principal weakness of the conventional audiometric technique for young children lies in its dependency upon purely subjective responses and the conscious cooperation of the child. Frequently, he cannot be made to understand or comprehend what he is supposed to do. This is especially true if the child has a language problem. It is apparent, moreover, that to the young child, pure-tone stimuli tend to be meaningless, uninteresting, and easily ignored. His attention span is short. Having him remain still with minimal movement for as long as half an hour is virtually impossible.[2] Furthermore, the need has been recognized for a reliable means of testing and treating children younger than 5 years of age. An early differential diagnosis can facilitate immediate treatment and therapy. It also enables one to obtain a more satisfactory social adjustment of the patient. As Myklebust[3] points out, a child with a hearing loss whose problem is not diagnosed or is incorrectly diagnosed will undergo undue hardship. He may be treated as mentally deficient, asphasic, or emotionally disturbed. Thus, he will suffer not only from his deafness, but he will be penalized further by being misunderstood. This may be prevented by an early accurate diagnosis which is followed by appropriate therapy. Psychological and personality problems consequent to auditory impairment may also be reduced by prompt diagnosis and treatment.

In addition, early diagnosis can help to facilitate optimum therapeutic results. Remedial measures can then be introduced at an early period of life, at a time when the child is most amenable and responsive to such corrective and rehabilitative procedures. Thus, if the best prognostic potentialities of the young patient are not to be abandoned, an accurate diagnosis should be made at the earliest time possible.

The need for testing young children, as well as others who cannot or will not give reliable audiograms by the finger-raising technique, has resulted in the attempt to develop new hearing tests which are more

Submitted for publication July 24, 1957.

This article is based on a dissertation submitted in partial fulfillment of the requirements for the Ph.D. degree at the University of Florida and the M.D. degree at Western Reserve University School of Medicine. The facilities for testing were provided by the Cleveland Hearing and Speech Center.

objective in nature. It is necessary to devise a test which is sufficiently objective so that observations are made which are independent from the statements of the patient.[4]

Responses which were more objective than those obtained with conventional pure-tone finger-raising audiometry were obtained when the conditioned psychogalvanic skin response was applied to acoustic stimuli. Hardy, Bordley, and Pauls[5,6] developed the use of the psychogalvanic skin response as a clinical instrument to measure auditory acuity. This technique is commonly called "PGSR" or "GSR" audiometry and is also known as the Electrodermal Response (EDR). The principle behind the technique is relatively simple. Changes in skin resistance in response to acoustic stimuli are used to indicate hearing acuity. A pure tone (conditioned stimulus—CS) is presented to the subject and is followed by a slight electric shock (unconditioned stimulus—UCS). The electric shock results in a change in skin resistance. With the repetition of this cycle, conditioning occurs and the change in skin resistance (now the conditioned response—CR) will occur when the tone alone is presented and the shock is omitted. Thus, observation of the conditioned response indicates that the subject heard the tone. Whether or not the tone must be perceived in the consciousness of the individual in order to yield such a response or whether it may result from a synapse below the conscious level is not fully known. Experimental evidence is wanting in this area.

The proper administration and interpretation of PGSR audiometry can be extremely difficult. It requires a skilled specialist for operation of the equipment and an assistant for the equally delicate task of preparing and handling the child during the procedure. The technique requires that the conditions of the testing situation be controlled with great care.[5]

The testing of children by means of PGSR audiometry, even when one takes all possible precautions, offers special difficulties. Although it does not rely upon the subject's voluntary response to the tone, it is hampered by the difficulty which one may have in differentiating responses which are associated with hearing the tone from those which are chance variations in skin resistance or responses to other stimuli.[7] It is for this reason that the objectivity of PGSR audiometry has been questioned.[8] Unless the audiologist is highly trained, there may be some uncertainty in distinguishing the PGSR's resulting from hearing the acoustic stimuli from the PGSR's following other stimuli.[8] However, if skilled and careful, the audiologist usually is able to distinguish the responses with a high degree of significance.[8] He is able to prevent a simple transfer of "subjectivity" from the subject to the audiologist.

The PGSR may be triggered by many stimuli—both internal and external. Body and limb movements, deep breaths (motor responses), emotional thought processes, startle reactions, tactile, visual, and acoustic stimuli are examples of stimuli which may cause a galvanic skin response or fluctuations in the galvanic readings.[8] Still other conditions may cause variation or interference with accurate interpretation. They include items such as instrumentation, type and placement of electrodes, and the kind, quality, and amount of electrode jelly.[8] Under fairly well-controlled conditions with an experienced experimenter using the proper equipment, most of these extraneous stimuli may be controlled. However, limb and body movements may prove to be particularly troublesome.

Accurate interpretation of responses is even more difficult with children who have additional motor problems such as cerebral palsy, athetosis, petit mal or some other type of cerebral degeneration or dysfunction resulting in motor impairment.[5] Especially in these cases, any excessive movement on the part of the child will register as a change in skin resistance level and may easily be confused with the conditioned response which is sought. A high degree of

motor involvement in a patient may produce such erratic movement of the galvanometer that it renders testing with the PGSR virtually impossible. Even with less severe involvement, a discernible conditioned PGSR and a reliable estimate of the child's hearing acuity may be extremely difficult to obtain. In some instances, the mere lack of cooperation on the part of the child may render the test useless, since passive cooperation is necessary. Thus, normally active young children as well as hyperactive patients with neuromotor disturbances cannot be tested effectively by PGSR audiometry because their activity interferes with the correct interpretation of the galvanic responses. The situation may become almost paradoxical in that the technique becomes more ineffective in those cases where it is most needed. Therefore, a means of controlling or reducing hyperkinetic, or at times just normal, juvenile muscular activity is needed and would prove of great assistance to the clinical testing of young children by means of PGSR audiometry. Reduction of limb and body activity must be achieved without, at the same time, affecting hearing thresholds. Thus, barbiturates have not proved to be satisfactory.[5] Nevertheless, effective premedication which does not effect hearing thresholds would not only facilitate the audiometric testing of normally active children, it would enable accurate evaluation of those children who are now denied the benefit of the test by the very nature of their pathological handicap.

Specifically, a medication is needed which will sedate and calm a child, reduce interfering motor activity but not put him to sleep, not be toxic, and, in particular, must not reduce his hearing acuity or inhibit his conditioned galvanic skin responses. The need for such medication has been long recognized,[5] especially for the infants and very young children since, in addition to other problems, they can be uncommonly difficult to condition.[9]

The use of sedating and tranquilizing premedication in audiometry should help to relax the patient and reduce the time necessary to establish rapport and confidence. This fact has already been recognized by the psychiatrist in his use of the tranquilizing drugs when dealing with disturbed patients.[10] From the very practical aspect of clinic administration, Hardy and Pauls[5] point out that such medication would serve as a boon in speeding up the testing of a large clinical load.

It would seem that when some of the extraneous variables which affect the PGSR audiometry are controlled, this method may open new horizons in the still relatively unexplored frontier of objective audiometry, and may help to distinguish language and communication disorders from peripheral deafness.[1]

Mephenesin Carbamate

Mephenesin carbamate (Tolseram)* is classified as an orally effective, centrally acting, skeletal muscle relaxant.[11] Producing muscular relaxation without concordant loss of consciousness or cerebral depression, it has a tranquilizing or sedative effect. It has been demonstrated to have a longer action than the parent compound, mephenesin, as exemplified by mean paralyzing doses on white mice and by sustained blood levels. The prolongation of action, as might be expected, has been attributed to the fact that, unlike its predecessor, it has no terminal hydroxyl group, and therefore is not rapidy oxidized to the inactive form, β-toloxylactic (myanesic) acid.

The action of mephenesin carbamate is superficially similar to that of curare in that skeletal muscular relaxation occurs. Unlike curare, however, it causes a selective depression of the transmission of neural reflexes through the spinal cord rather than resulting in a blockage of acetylcholine at the myoneural junction. It particularly depresses transmission through the internuncial neurons.[12] The chief sites of action appear to be subcortical, in the brain stem,

* Supplied for this study by E. R. Squibb & Sons, New York.

at the level of the thalamus and below in the spinal cord. Polysynaptic spinal reflexes appear to be more readily inhibited than monosynaptic reflexes, and supraspinal facilitation and suppression of these spinal reflexes are abolished by the drug.[10] As evidence against any cortical effects, clinical doses produce no detectable changes in the normal human electroencephalogram.[12]

Mephenesin carbamate is readily absorbed via the oral route but may be administered intravenously. It is quickly distributed uniformly throughout most of the body tissues. As a general rule, the concentration of the free drug in the plasma correlates directly with the extent of the pharmacological effects produced.[10]

Mephenesin carbamate has been studied both in laboratory animals and in man. Mice and rats given oral doses of the drug ranging from 650 to 1500 mg. per kg. weight daily for a period of 13 weeks demonstrated no inhibition of growth, no hematological changes or any significant pathology. Dogs receiving mephenesin carbamate for 26 weeks appeared normal with respect to growth, hematology, blood chemistry, and liver and kidney function. All organs were found to be normal at autopsy.[13]

Reports of toxicity following administration of the parent drug (mephenesin) indicated that any untoward reactions occurred only after massive doses were given intravenously over a long period of time. No such reactions resulted from the oral route of administration in which absorption is gradual.[14] The toxicity of mephenesin carbamate was found to be even less than that of mephenesin.[13] In view of the fact that no toxicity has been reported following the oral route for mephenesin carbamate, the relative safety of the drug may be assumed, the likelihood of any untoward reactions being remote. There are no contraindications to the use of mephenesin carbamate.[13]

The fact that mephenesin carbamate is a safe, nontoxic, orally effective sedative and muscle relaxant which does not affect the cranial nerves but whose site of action is at the level of the brain stem and below, might make it a likely candidate for premedication in PGSR and other types of audiometry. Mephenesin carbamate is available in 0.5 gm. tablets and as a pleasant-tasting peppermint-flavored pediatric suspension for infants and young children.

Before mephenesin carbamate, or similar agents with the same site of action, are employed, it would be desirable to investigate the effects of such medication upon hearing thresholds, determined by conventional audiometric methods as well as by PGSR audiometry. The latter aspect would indicate indirectly any possible effects of the drug on either conditioning or the psychogalvanic skin response. This would be especially desirable in view of the potential benefit that a medication such as mephenesin carbamate might offer in promoting the successful application of PGSR audiometry to infants and young children. Conversely, in the event that either hearing thresholds or the conditioned psychogalvanic skin responses are affected by such premedication, the advisability of utilizing such a drug in connection with audiometry would be open to question. Should mephenesin carbamate not cause any statistically significant variation in hearing thresholds as determined by both of these methods, then further studies with mephenesin carbamate, tranquilizers, and similar preparations might be undertaken.

The purpose of the present study is to investigate the effects of mephenesin carbamate on normal adult hearing thresholds as determined by conventional pure-tone audiometry and by conditioned PGSR audiometry.

Instrumentation and Procedure

The question to be answered by the study is whether the administration of mephenesin carbamate prior to testing will affect the

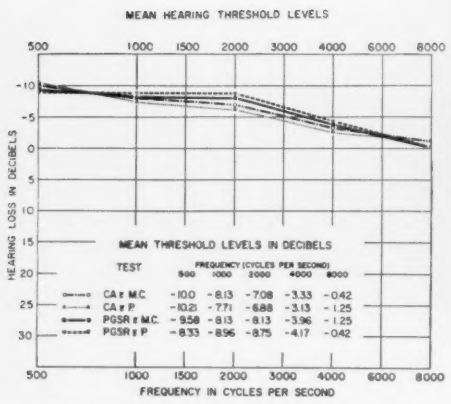


Fig. 1.—Mean hearing threshold levels.

thresholds of hearing measured either by conventional pure-tone audiometry or by conditioned PGSR audiometry.

The tests were administered at the Cleveland Hearing and Speech Center in a sound-treated room with a low ambient noise level. This room was adjacent to a control room in which the testing equipment was placed. A large double plate glass window over the control panel provided a view of the testing room. The subject could be observed directly through this window as well as by way of a mirror on the opposite wall of the testing room.

The subjects were seated in a comfortable armchair, and the electrodes were applied to the proximal interphalangeal joints of the first and third fingers of each hand as well as on the wrist of the left hand. A standard electrode jelly, Redux electrocardiographic jelly, was used. The faradic electric shock was received by the subject in the left third finger. The galvanic skin response was picked up and measured from the right first and third fingers.

A wooden board designed to fit over the arms of the chair and extend as a table in front of the subject can be used with young patients as a place on which to rest their arms and hands and on which to place toys or similar distracting paraphernalia. For very young children, it is advisable to use electrodes which attach to the feet rather than to the hands or fingers.

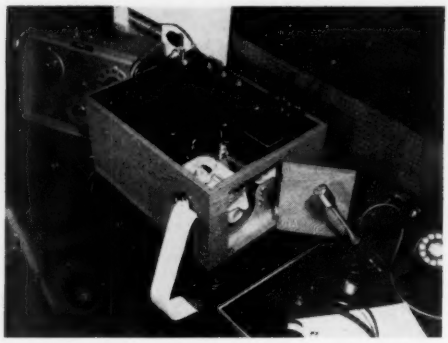


Fig. 2.—View of the equipment. The Beltone Audiometer on the left connects directly to the Grason-Stadler Psychogalvanometer and is controlled automatically by an electronic timing mechanism in the Psychogalvanometer. A microphone and earphones for intercommunication with the subject in the testing room are in the lower right-hand corner.

A Grason-Stadler GSΔR automatic psychogalvanometer with a direct writing, heat stylus, Sanborn recorder plus a coupled Beltone 10-A audiometer were employed. Although the duration of and the interval between the conditioned (CS) and unconditioned (UCS) stimuli could be varied, once the CS was initiated, the CS and UCS were automatic and were electronically controlled. The duration of the CS (2.0 seconds) and UCS (0.75 seconds), as well as the interval between them (0.5 seconds), was kept constant throughout the experimentation.

The audiometer calibration was checked regularly with a standard 6 cc. coupler, the Bruel and Kjaer Artificial Ear. After preliminary testing, because of the excellence of the subjects' hearing and the low threshold values obtained, it was found necessary to add a 20 db. T-pad (12 ohm resistance) in the earphone line. Thus, the limit of the audiometer was extended from -10 db. to -30 db. Since no subject was found to have a threshold value below -20 db. at any frequency tested and most thresholds were considerably above this level, this pad proved to be very adequate. Both the conventional pure-tone test and the PGSR evaluation were conducted with use of the same Beltone audiometer.

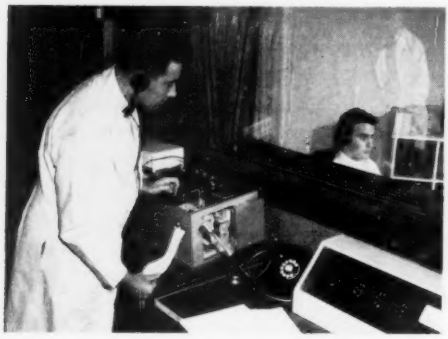


Fig. 3.—Conducting the test. The subject remains in full view of the experimenter throughout the test. Although this is not necessary for PGSR audiometry, it is considered desirable to observe the subject after he has received medication, even when there is little likelihood of any untoward reaction occurring.

Twenty-four adult male subjects participated in the study. Their ages ranged from 21 to 28 years with a mean age of 23.79 years and a median age of 24. The distribution of ages was flat, showing bimodal peaks at 22 and 24 years of age. All the subjects were full-time medical students at Western Reserve University School of Medicine who, to their knowledge prior to testing, had normal hearing. The subjects were selected at random from a group of 320 students at the Medical School. They were in good health at the time of the investigation.

Each subject received 2.0 gm. of mephenesin carbamate or an equal amount of lactose placebo approximately one-half hour before each of four tests. The drug and placebo were both triturated and encapsulated so that no clues were available to the subject such as taste, color, or odor. A half-hour between the time of taking the capsules and commencing the tests allowed for maximum absorption of the drug and for peak plasma levels during the test.[11] All the capsules were swallowed with fruit juice in order to lessen the possibility of any gastrointestinal disturbances following the ingestion of mephenesin carbamate. Throughout the entire procedure, both the experimenter and the subjects were unaware of the capsules' contents.

When selected, the subjects were aware of the fact that their hearing would be tested by two methods and that they would be given capsules to swallow prior to each of four tests. They knew that these capsules would contain either a muscle relaxant or a placebo. Having the subject cognizant of the possibility that he might be getting a placebo tended to reduce the tendency toward overreaction. The knowledge that a placebo was involved in the investigation tended to yield a more conservative type of introspection, which aided the subjects in subjectively judging the effects of the medication.[15]

Each of the 24 subjects received 4 tests so that a total of 96 tests were administered in the study. The four tests that each subject was given were as follows:

1. Conventional audiometry with mephenesin carbamate (C. A.—M. C.)
2. Conventional audiometry with placebo (C. A.—P)
3. PGSR audiometry with mephenesin carbamate (PGSR—M. C.)
4. PGSR audiometry with placebo (PGSR—P)

A counterbalanced order of tests was achieved by a Latin square arrangement.[16] The tests for each subject were administered one week apart at the same time of day, whenever possible, in order to reduce practice and fatigue effects which might not be cancelled by the counterbalanced order.

When tested by conventional pure-tone audiometry, the subject was told to raise his finger as soon as he heard a tone and to keep his finger raised as long as he could hear the tone. With PGSR audiometry, the subject was told that all that he would be required to do for the test was to relax and sit quietly with as little body and limb motion as possible. He was informed that he would hear tones which might or might not be followed by a slight electric shock. There would be no overt response required of him. In all cases, the subjects were observed from the darkened control room and could be seen directly through the window or indirectly via a mirror in the testing room.

Threshold values were obtained for 500, 1000, 2000, 4000, and 8000 cps. In the test procedure, the subject was presented with a 1000 cycle tone at an intensity of 40 db. above the normal hearing threshold level (0 db.). This initial unreinforced CS was given to determine the state of conditioning at the commencement of the test. The subject was then conditioned to this 1000 cycle tone at an intensity level of 40 db. Although Doerfler and McClure [17] found that a frequency of 500 cps and an intensity level of 80 db. were ideal for conditioning, they also showed that a 1000 cps tone could be used quite adequately. They conditioned their subjects to a 1000 cycle tone at a level of 70 db. above normal threshold. In the present study, the subjects were conditioned to 1000 cps because, in an evaluation of a patient's hearing, if extenuating circumstances permit only one frequency to be checked, it was felt that from the point of view of speech range, the most desirable frequency to be investigated would be 1000 cps.

The tone (CS) was presented for 2.0 seconds' duration. The shock (UCS) was given 0.5 second after the onset of the tone. The shock duration was 0.75 second, and its intensity was varied from 0.15 ma. to 2.0 ma., depending upon the individual subject's sensitivity and shock threshold. An attempt was made in each case to adjust the shock level to approximately three just noticeable differences (JND's) above the subject's threshold for the shock. The same shock intensity was used throughout the test for each subject.

Stimuli, both CS and UCS, were presented according to a random schedule.[16] The time interval between successive tones was also varied randomly with a minimum of 20 seconds and a maximum of 90 seconds. These values are within the limits suggested by Stewart.[8]

Consistent with the findings of Hovland [18] and of Meritser and Doerfler,[19] Doerfler and McClure [17] found that resistance to extinction of the conditioned response is significantly greater with 40% to 60% reinforcement than with 100% reinforcement of the conditioned stimulus with the unconditioned stimulus. In accordance with these findings, the subjects in this study were conditioned with use of partial (60%) reinforcement.

Although no basis has been found for assuming that there is a difference between varied and constant intensity of stimuli as affecting the acquisition or extinction of the conditioned response,[19] the intensity level was kept constant at 40 db. during the conditioning process of this investigation. The intensity level was varied only after the conditioned response was established and an estimate of the threshold was being made using the method of limits with ascending and descending series.

Basal levels of skin resistance ranged from a low of 10,000 ohms to a high of 180,000 ohms. The mean value of the maximum resistance for all subjects was 61,670 ohms when mephenesin carbamate was administered and 63,750 when the placebo was given. The mean values of the minimum resistance were 43,130 ohms and 45,000 ohms with the administration of mephenesin carbamate and the placebo respectively. Differences in basal levels when the drug was given as compared with those measured when the placebo was used were not statistically significant.

The criterion for conditioning was a minimum of three responses to three unreinforced tones. The total time required to establish conditioning ranged from 1 minute and 30 seconds to 1 hour and 10 minutes. In three tests, no conditioned responses were evident after 1 hour and 45 minutes. The attempt was abandoned after 150 trials. Two of the instances occurred with the same subject, involving both his PGSR tests. It was not found possible to condition him under the time restrictions of the experiment. In considering all the responses, the special criteria outlined by Doerfler and McClure [17] were followed. As basic criteria for judging whether or not a

deflection was a response, a minimum of 5 mm. in height and a 45 degree slope were used.[17]

Thus, each subject was presented with a 1000 cycle 40 db. tone which was reinforced randomly by shock 60% of the time. When a conditioned response was obtained, the intensity level of the tone was gradually reduced in a descending series until a conditioned response no longer appeared. Then the tone intensity was gradually increased, in an ascending series, until the conditioned response appeared again, indicating that the subject had heard the tone at that intensity.

After the threshold for 1000 cps was established, a 2000 cps tone was presented at 40 db. The method of limits was repeated with occasional reinforcement when the responses became very low in amplitude. Just enough reinforcement was given to maintain conditioning. After the threshold value was ascertained for 2000 cps, the process was repeated at 4000, 8000, and finally at 500 cps. A CS of 1000 cps at 40 db. (reference tone) was presented after each threshold value was obtained as a check on the level of conditioning. When necessary, reinforcement with shocks was given.

Sufficient generalization occurred so that the initial conditioning process achieved with the 1000 cps 40 db. tone was strong enough to carry over to the other frequencies and intensities.

Little or no difficulty was encountered in distinguishing galvanic skin responses to the tone from random variations in skin resistance. The latter were very few in number as might be expected in testing cooperative adult subjects. This lack of difficulty in recognizing the PGSR when testing adults is consistent with the literature. Stewart [8] found an extremely high degree of certainty in distinguishing conditioned PGSR's from responses to extraneous stimuli. He found less than one chance in a thousand of an error in distinguishing the PGSR from other responses when testing cooperative adults.

No attempt was made to do any experimental extinction of the conditioned responses. No instances of untoward reactions were experienced with the administration of either mephenesin carbamate or the lactose placebo in this experiment.

Analysis and Evaluation of the Data

To test the hypothesis proposed in this study by an appropriate statistical method, an analysis of variance was used with two criteria of classification.[20] In the analysis, the variation between groups was compared to the variation within groups.

Mean threshold values obtained by the four tests at each of the five frequencies investigated appear in Figure 1.

The results of the analysis of variance at each frequency show that there were no differences in thresholds which cannot readily be attributed to chance variation.

In the light of these findings, the hypothesis that there is no significant difference between adult auditory thresholds after the administration of mephenesin carbamate or a lactose placebo as tested by both conventional and PGSR audiometric techniques may not be rejected. Mephenesin carbamate causes no statistically significant changes in hearing thresholds, whether they are measured by conventional or PGSR audiometry.

Comment and Conclusions

In this experiment, 24 normal hearing, adult male subjects were tested in four ways, using a Latin square counterbalanced order. Each subject's hearing thresholds at 500 cps, 1000 cps, 2000 cps, 4000 cps, and 8000 cps were estimated by conventional pure-tone audiometry following the administration of mephenesin carbamate or a lactose placebo, as well as by PGSR audiometry also following the administration of mephenesin carbamate or suitable placebo. A statistical analysis of variance of the quantitative data indicated that mephenesin carbamate has no statistically significant effect upon hearing thresholds as determined by either PGSR or

conventional pure-tone audiometry. Furthermore, no significant differences in thresholds were found whether measured by conventional pure-tone finger-raising technique or by PGSR audiometry.

In the light of these findings, further investigation might be made into the feasibility of employing mephenesin carbamate, or similarly acting pharmaceutical preparations, as sedative and tranquilizing premedication for PGSR and other types of audiometry as the demand arises for facilitation of testing.

Since mephenesin carbamate may be used without interfering with or significantly affecting hearing thresholds, a way is paved for further inquiry as to the effectiveness of the drug in reducing the activity of normal children and the hyperactivity of patients with neuromotor disorders.

The eventual goal to be achieved is the selective administration of this or similar drugs by the otologist as premedication in clinical audiometry in order to facilitate the testing of those patients whose hearing thresholds cannot be evaluated satisfactorily by existing methods because of normal or abnormal but interfering motor activity.

Summary

The purpose of the investigation is to study the effects of mephenesin carbamate on normal hearing thresholds as determined by two methods: the conditioned psychogalvanic skin response (PGSR) and conventional pure-tone audiometry. The rationale for the investigation stems from the inadequateness and unreliability of conventional means of testing the hearing of infants, children, malingerers, and persons with psychogenic deafness. The use of PGSR audiometry has been helpful but has been ineffective in cases where it is most needed. Normally active young children and hyperactive patients with neuromotor disorders cannot be tested satisfactorily by PGSR audiometry because their activity interferes with the correct interpretation of the galvanic responses. Some means of reducing limb and body motor activity without, at the same time, affecting hearing thresholds is needed. Mephenesin carbamate, a muscle relaxant, is investigated as possibly fulfilling these requirements. It acts subcortically, at the level of the thalamus, in the brain stem, and below in the spinal cord, specifically on the internuncial neurons. In view of this site of action, if it could be shown that mephenesin carbamate did not affect hearing thresholds, investigation might be made as to its utilization as premedication to facilitate hearing testing.

To demonstrate that mephenesin carbamate does not affect hearing sensitivity, 24 normal hearing, adult male subjects, with an age range of 21 to 28 years and a mean age of 23.79 years, are tested in four ways using a Latin square counterbalanced order. Each subject's hearing thresholds at 500 cps, 1000 cps, 4000 cps, and 8000 cps are estimated by conventional and PGSR audiometry following the administration of mephenesin carbamate or a suitable lactose placebo. A Grason-Stadler Psychogalvanometer with an interconnected Sanborn direct-writing recorder is used to measure the PGSR, along with a Beltone audiometer. A statistical analysis of variance of the quantitative data leads to the following conclusions:

1. Mephenesin carbamate has no statistically significant effect upon hearing thresholds as determined by PGSR audiometry.

2. Mephenesin carbamate has no statistically significant effect upon hearing thresholds as determined by conventional pure-tone audiometry.

3. There are no statistically significant differences between hearing thresholds of the same persons measured by conventional pure-tone or PGSR audiometry.

Western Reserve University School of Medicine.

REFERENCES

1. Goldstein, R.; Polito-Castro, S. B., and Daniels, J. T.: Difficulty in Conditioning Electrodermal Responses to Tone in Normally Hearing Children, J. Speech & Hearing Disorders 20:26 (March) 1955.

2. Keaster, J.: Quantitative Hearing Tests for Young Children, Volta Rev. 50:465 (Sept.) 1948.

3. Myklebust, H. R.: Auditory Disorders in Children, New York, Grune & Stratton, Inc., 1954.

4. Kobrak, H. G.: Objective Audiometry, A. M. A. Arch. Otolaryng. 65:26 (Jan.) 1957.

5. Hardy, W. G., and Pauls, M. D.: The Test Situation in PGSR Audiometry, J. Speech & Hearing Disorders 17:125 (March) 1952.

6. Bordley, J. E., and Hardy, W. G.: A Study in Objective Audiometry with the Use of a Psychogalvanic Response, Ann. Otol. Rhin. & Laryng. 58:759 (Sept.) 1949.

7. Galloway, F. T., and Butler, R. A.: Conditioned Eyelid Response to Tone as an Objective Test of Hearing, J. Speech & Hearing Disorders 21:47 (March) 1956.

8. Stewart, K. C.: Some Basic Considerations in Applying the GSR Technique to the Measurement of Auditory Sensitivity, J. Speech & Hearing Disorders 19:174 (June) 1954.

9. Goldstein, R.; Ludwig, H., and Nauton, R. F.: Difficulty in Conditioning Galvanic Skin Responses: Its Possible Significance in Clinical Audiometry, Acta oto-laryng. 44:67, 1954.

10. Goodman, L. S., and Gilman, A.: The Pharmacological Basis of Therapeutics, Ed. 2, New York, The Macmillan Company, 1955.

11. Dresel, P. E., and Slater, I. H.: Observations on the Pharmacology of Mephenesin Carbamate, Proc. Soc. Exper. Biol. & Med. 79:287 (Feb.) 1952.

12. Henneman, E.; Kaplan, A., and Unna, K.: A Neuropharmacological Study of the Effect of Myanesin (Tolserol) on Motor Systems, J. Pharmacol. & Exper. Therap. 97:331 (Nov.) 1949.

13. Thomas, B. G. H.; Poutsiaka, J. W.; Kulesza, J. S., and Linegar, C. R.: Toxicity and Blood Level Studies on 3-(o-Toloxy)-2-Hydroxypropyl Carbamate (Tolserol Carbamate), J. Pharmacol. & Exper. Therap. 110:48 (Jan.) 1954.

14. Godman, H. E., and Adriani, J.: Management of Patients with Tetanus: Some Clinical Experiences with Various Muscle-Relaxing Agents, J. A. M. A. 141:756 (Nov. 12) 1949.

15. Drill, V. A., Editor: Pharmacology in Medicine, New York, McGraw-Hill Book Company, Inc., 1954.

16. Fisher, R. A., and Yates, F.: Statistical Tables for Biological, Agricultural and Medical Research, Ed. 4, Edinburgh, Oliver & Boyd, Ltd., 1953.

17. Doerfler, L. G., and McClure, C. T.: The Measurement of Hearing Loss in Adults by Galvanic Skin Response, J. Speech & Hearing Disorders 19:184-189 (June) 1954.

18. Hovland, C. I.: The Generalization of Conditioned Responses: IV. The Effects of Varying Amounts of Reinforcement upon the Degree of Generalization of Conditioned Responses, J. Exper. Psychol. 21:261 (Sept.) 1937.

19. Meritser, C. L., and Doerfler, L. G.: The Conditioned Galvanic Skin Response under Two Modes of Reinforcement, J. Speech & Hearing Disorders 19:350 (Sept.) 1954.

20. Snedecor, G. W.: Statistical Methods, Ed. 4, Ames, Iowa, The Iowa State College Press, 1946.

Case Reports

Congenital Anomaly of Nasal Structure

MICHAEL S. HOROWITZ, M.D., London

Description

A patient, age 27, consulted me about a congenital deformity of his nose involving the osteocartilaginous structure of the nasal pyramid. On examination the shape of the nose resembled a matchbox. The width of the root of the nose was 4 cm., the same as that of the tip. The bridge of the nose presented a large depression starting at the level of the glabella, where the depression was very slight, and became deeper toward the tip of the nose, where the depression passed between the medial crura of the lower cartilages. The angles of the lower cartilages of the nose were separated by this depression. The bridge of the nose was limited on each side by a ridge beginning at the glabella and finishing at the angle of the lower cartilages. The length of the nose was 5.2 cm. The nostrils were asymmetric and the left was larger than the right, owing to the greater size of the left lower cartilage. The left nostril was slightly directed upward and outward. The skin of the bridge appeared to be thinner than that of the rest of the nose. The septum was very broad, not deflected, and the approximate breadth was 1 cm. There was no evidence of any other deformity of the head. The right side of the nose was slightly longer than the left side. The distance between the eyes appeared to be much wider than normal.

There was no impairment of nasal breathing.

Accepted for publication, June 26, 1957.

Presented at the Sixth International Congress of Otolaryngology, Washington, D. C., May 9, 1957.

X-Ray Examination

The x-ray showed a faulty development of the bony structure of the nasal bones with absence of ossification.

The patient consulted me because of the inferiority complex which he had developed in childhood and which persisted. At school the children used to bully him, calling him "flat nose," "squash nose," and so on. He used to take refuge from the aggressive attitude of the children at school and only at home with his parents, brothers, and sisters did he feel relaxed and happy.

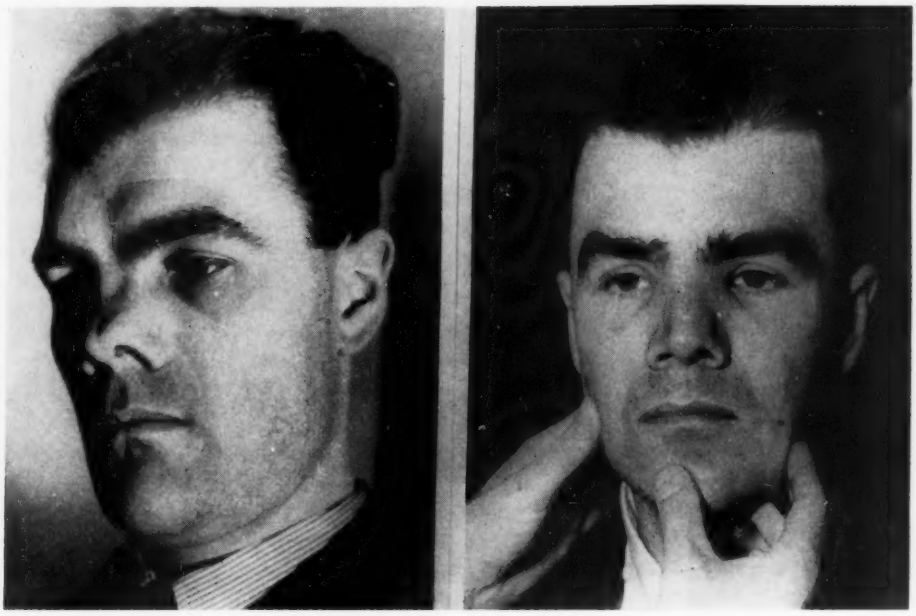
During the war he served in the Navy and was in contact with new people who seemed kindly disposed toward him; nevertheless, after the war he reverted to his former reserved attitude toward life.

Heredity.—There are no congenital deformities in parents or any other members of the family.

I decided to perform a corrective operation.

Operative Technique

Despite the fact that most of the nasal plastic operations are performed from within, it is advisable in certain cases to approach it externally because the inside operation does not permit a precise recognition of the deformities of the internal structures of the nose. The outside operation gives a clear picture of the deformities and makes a reconstruction of the nose much easier, and this technique also helps in the correction of the apparent increased distance between the eyes (hypertelorism).



Before (left) and after (right) corrective surgery.

The technique consists in making a long skin incision starting midway between the eyes and ending at the upper border of the upper lip. The incision should pass between the medial crura of the lower cartilages and the columella. Precautions should be taken not to make an incision in the longitudinal axis of the nose, but of the longitudinal axis of the face, and all the structures of the nose ought to be directed toward this longitudinal axis of the face. After having undermined all the soft tissues of the nose one should expose the arch of the nose as well as the ascending nasal processes of the maxillary bones. Once this area is exposed, one makes two parallel incisions at the level of the two ridges of the bridge of the nose limiting the depression, and with chisel and hammer the cartilaginous septum should be removed in block, leaving behind only the perichondrium and a very thin layer of cartilage on each side of the septum.

To prevent injury which may result in perforation of the perichondrium and the mucous membrane one should protect these structures by introducing the little finger into the nasal cavity while performing the incision of the septal block. Once the block of cartilage is removed one finds a very deep groove between the two ridges. One should now divide the ascending processes of the maxilla with a file or chisel and displace them toward the midline of the face in order to reduce the width of the nose. One should also separate the medial crura of the lower cartilages, and all the subcutaneous tissue between them should be removed to reduce the width of the tip of the nose.

At the same time, part of the longer crura should be excised in order to reduce the size of the larger nostril. After all the cartilages are mobilized toward the midline, they are fixed in position with a few points of absorbable surgical suture. At the same time it is advisable to put in a few stitches at the level of the middle crura and at the angles of the lower cartilages. After fixing the bony and cartilaginous structures in position one excises the surplus skin. It is advisable to do a large excision of the skin at the level of the glabella in order to bring the

inner ends of the eyebrows nearer together to give to the distance between the eyes the appearance of less width. After excision of surplus skin it is advisable to do an intradermic suture going alternatively from one side to the other of the incision. One should use very fine silk or a very fine steel wire, which is much easier to remove after four or five days. This intradermic suture ought to be reinforced by a few tension sutures.

Dressing.—Cover by a small piece of dry gauze and keep in position by a small support made of plaster of Paris which is fixed with a few bands of Elastoplast.

Etiology

The cleft nose, or the "doggen nose" as the Germans call it, is one of the rarest congenital malformations of the nose. There are very few cases which have been described since the end of the last century and we shall find that rarely has a surgeon with a long professional career had the opportunity to see more than one case in his lifetime. The first case of such a congenital malformation of the nose was described by Witzel in 1882. After him Lexer, Nasse, and Kredel described a few isolated cases of the same type. From the literature of the different authors which we mentioned above, it appears that there are two different opinions as to the cause of this embryological malformation of the nose. According to Witzel, it is due to an intracanial pressure, while Lexer, Nasse, Kredel, and other authors consider amniotic faults to be responsible for the formation of the cleft nose.

The number of such cases in living children is very small but a few more cases have been described of cleft noses in the fetus which were not viable. It may be that the latter cases are more frequent than we realize but they escape our attention for the reason they are associated with more important congenital deformities and we do not pay any attention to the relatively minor deformity of the nose. If we consider the small number of these deformities of the nose, and the different aspects of these deformities, we realize the difficulty in establishing a standard operation for these cases.

According to Witzel the cleft nose is due to a force deriving from inside and separating the different parts of the nose which exist in pairs. In these cases there should, at the same time, exist a cleft of lip and hard palate. On the other hand, according to Nasse the cleft nose is caused by some power which comes from outside and which should be considered as the cause of the trouble in the development of the normal nose. After Trendelenburg and von Bramann we ought to consider that the congenital median fistula of the nose and the dermoids present the very last formation of the cleft nose. Von Bramann thought that the development of the nasal dermoids can be explained only through the relation of the head of the fetus to the amniotic membrane. And he thinks that such amniotic adhesions are responsible for the formation of the cleft nose.

Witzel concluded from the observations that it was quite difficult to explain this malformation because the cleft is limited to a part of the body which does not exist in pairs but singly. The other clefts in the region of the face, the neck, and the body are considered in such a way that the union of the parts of each side are impaired in their approachment, which can easily be compared with the different phases of the development of the fetus.

A fetal cleft of the frontal process has not been described. In most of the cases the original factor is not to be found in a force deriving from inside the skull, but it is quite evident that the initial factor comes from outside, and we shall find different abnormalities of the amniotic pressure coming from amniotic faults or due to some adhesions of the amniotic membrane. This last opinion seems, according to Kredel, to be more applicable in most cases of different degrees of congenital malformation, from the smallest to the most important ones. The theory of the increased intracanial

pressure (after Witzel) cannot be applied in the very superficial cases of cleft noses. From the moment that we free ourself from Witzel's theory that in the cleft noses there is always a defect of the frontal process, and we consider the cleft of the nasal pyramid as primary defect, then we shall have understood the cause of the congenital deformity of the nose. The nose is formed around the single frontal process, but, on the other hand, it is an organ which is built up by structure in pairs. The union of nasal elements growing toward the median line may be inhibited in the same way as we see it in the superior maxilla in the formation of the cleft palates. The degree of the malformation is in direct proportion to the area, the time, and the duration of the amniotic adhesions.

120 Harley St. (W. 1)

Nonchromaffin Paraganglioma of the Middle Ear

Report of Four Cases

THOMAS R. COX, M.D.; WALTER KROHN, M.D., and LUIS E. REMUS, M.D., Dallas, Texas

During the past three years we have seen at Baylor University Hospital four cases of nonchromaffin paraganglioma or carotid-body-like tumor of the middle ear. Up to 1951 only 18 cases had been reported under this name in the entire world literature. In 1953, however, J. C. Gaffney described one case of this tumor, reviewed the world literature, and compiled a list of 56 cases, many reported under different names, which he believed to represent this lesion. Since 1953 there have been many reports of this tumor, primarily in the literature of otology, laryngology, and rhinology.

Report of Cases

CASE 1.—A 46-year-old woman had developed an abscess of her right ear six years prior to this admission. The abscess had responded to antibiotic therapy, but afterwards she had had a slight diminution in hearing on this side. In 1953, five years after the abscess formation, she noted slight bleeding from the ear, but no pain. She was referred by her local physician to an otologist with a diagnosis of "granuloma of the middle ear." On examination, the external canal was open but the middle ear was filled with a red, vascular-appearing lesion which bulged outward the tympanic membrane. In July, 1954, six years after the onset of her disease, a radical mastoidectomy was performed. At surgery the tumor was highly vascular, and extensive bleeding was encountered. The postoperative course was uneventful. At the present time, three years after surgery, there is no evidence of recurrence of the tumor.

Gross Description.—The specimen consisted of a soft, pedunculated mass 17×9×8 mm., covered by a wrinkled gray epithelium. The cut surface was red-gray and vascular (Fig. 1).

CASE 2.—A 31-year-old man had a long history of ear trouble dating back 11 years to 1944, at which time he had been rejected from the Army because of a perforated left tympanic membrane. Since then the man had seen several ear, nose, and throat specialists with the diagnosis of fungus disease, otitis externa, and impacted cerumen of the external ear. He had received two radiation therapy treatments, for which indications are not clear. Intermittently, he was seen by different doctors until 1951, when he experienced a severe hemorrhage from the left ear. At that time a tumor mass was found in the middle ear, which was thought to be hemangiomatous in type with secondary inflammatory reaction. Surgery was recommended but apparently refused. In 1953 cobalt irradiation was applied, and this resulted in retraction of the mass to the point that adequate cleansing could be done. After irradiation, however, the patient continued to have symptoms of chronic otitis media and progressive diminution of hearing. Surgery had been urged consistently during the interim and was finally performed in July, 1955. Except for excessive bleeding the operative procedure was simple, utilizing the usual approach to a radical mastoid operation in the modified Lempert technique. To the present time there has been no sign of tumor recurrence.

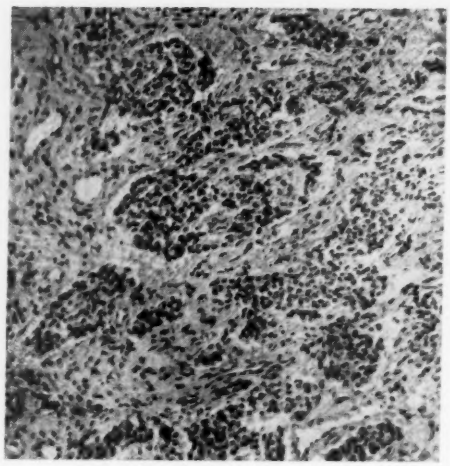


Figure 1; × 100.

Accepted for publication June 13, 1957.

From the Department of Pathology, Baylor University Hospital.

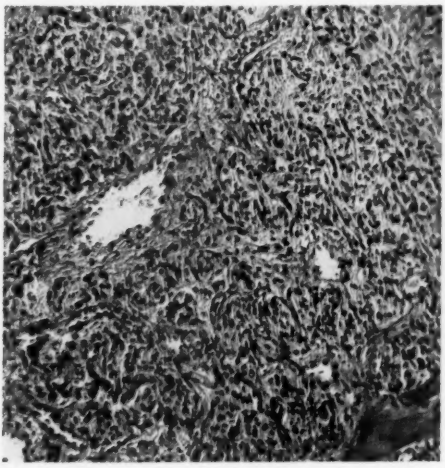


Figure 2; × 100.

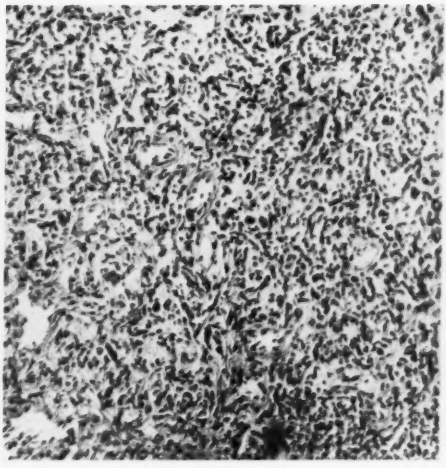


Figure 3; × 100.

Gross Description.—The specimen consisted of two oval, soft, pink-red portions of tissue measuring 3 mm. each. The cut surface was red and hemorrhagic (Fig. 2).

CASE 3.—A 35-year-old woman had complained of pain, pulsation, and progressive deafness in her right ear over a period of several years. In February, 1956, a firm nodular mass was found completely to seal the inner portion of the external canal and push the tympanic membrane ahead of it. This mass did not blanch on pressure. An x-ray examination showed normal mastoid cells. In April 1956 a modified radical mastoidectomy was performed for removal of the tumor mass. Again considerable bleeding was associated with the procedure. There has been no evidence of tumor recurrence to the present time.

Gross Description.—The specimen consisted of several portions of yellowish-tan, hemorrhagic, soft tissue from 2×2 mm. to 5×6 cm. (Fig. 3).

CASE 4.—A 52-year-old white woman had had deafness and dull aching sensation in the right ear for several years. In 1954 she had had a polypoid mass removed from her right ear. Two years later she developed a right facial paralysis associated with aching in the right ear and pain behind the right eyeball. On examination there was a complete right facial paralysis. The right ear showed a large lobulated smooth reddish-brown mass completely filling the external auditory canal. The tympanic membrane and middle ear were invisible. X-ray examination revealed slight clouding of the mastoid but no erosion. In November, 1956, an endaural tympanomastoidectomy was performed. The tumor was found to fill the attic and middle ear, extending backward into the antrum. Severe bleeding from the jugular bulb occurred after subtotal removal of the tumor. The hemorrhage was controlled by packing. Two weeks after surgery the pack was removed, and the tumor was seen to have completely eroded and destroyed the facial canal and facial nerve in its intratympanic portion. The middle ear portion of the tumor was removed. As soon as the anterior-inferior region was approached, severe bleeding occurred, forcing suspension of surgery. One week later the jugular vein was found to be subtotally collapsed, and when the pack was removed free bleeding occurred, which was not affected by temporary ligation of the common carotid artery. The bleeding was thought to arise from the inferior petrosal sinus. It was controlled by packing the bony anterior and inferior opening to the jugular bulb; then all evident tumor was

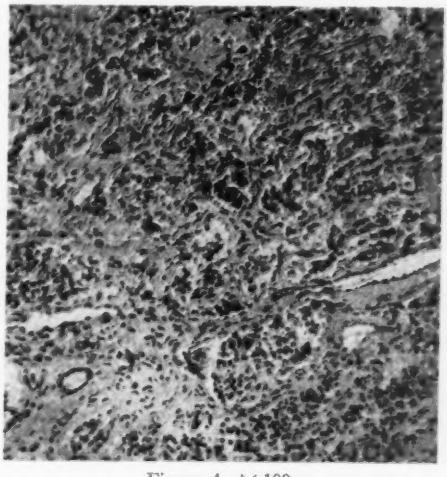


Figure 4; × 100.

removed. At the present time, the ear is completely healed, and no recurrence of the tumor is seen.

Gross Description.—The specimen consisted of three fragments of soft, friable, brownish-red tissue measuring in the aggregate 7 mm., together with two larger fragments of light brown firm tissue, 15×5 mm. each (Fig. 4).

Histological Features

All tumors were essentially similar with minor variations. The over-all picture was one of a rich network of sinusoids within which lay clusters of small regular, round, oval, or spindle cells. The nuclei were regular in outline and chromatin distribution. No mitoses were seen. Varying degrees of fibrosis and hemorrhagic necrosis were manifest. Reticulin stains clearly outlined the sinusoidal network surrounding the cell clusters. The variations of the tumor were consistent with those seen in the usual carotid-body tumor (Figs. 1 to 4).

Comment

In 1941, Guild described small glomus-like formations situated in the floor of the middle ear in the adventitia of the bulb of the jugular vein. Usually they lie in the dome of the jugular bulb immediately beneath the bony floor of the middle ear and near the ramus tympanicus of the glossopharyngeal nerve. Occasionally, two or more are present. These have been reported as the glomus tympanicus and jugulare. It is from these bodies that the nonchromaffin paraganglioma or carotid-body-like tumors of the middle ear are derived.

The tumor is commoner in women than in men in about a 3:1 ratio and occurs most commonly in middle-aged and older people. Of our four cases, three were women in the mid-30's and older.

The symptomatology is usually of long duration, as is seen in our cases, and is characterized by evidence of chronic infection, diminution in hearing, dull aching pains and occasional episodes of hemorrhage. On physical examination, the tumor is frequently mistaken for a pyogenic type of granuloma because of its extreme vascularity. It may be seen bulging the tympanic membrane and filling the middle ear, or may perforate the tympanic membrane and occlude the external canal as well. X-ray examination of the mastoid has proved of little help in determining the extent of this tumor. Occasionally involvement of such structures as the facial nerve may give rise to neurological findings, as was seen in our Case 4. Invasion of various portions of the petrous temporal bone, the ossicles, and the labyrinth have all been reported. In the case of Weille and Lane, the tumor extended forward through the Eustachian tube to present in the left nasopharynx. Dockerty in 1951 reported a case in which the tumor eroded the petrous and occipital bones to form part of the floor of the posterior cranial fossa. In Seiffert's case infiltration and thrombosis of the sigmoid sinus were seen. In all clearly substantiated cases, the tumor showed a marked local invasiveness, but in none has metastasis occurred.

Many references are made throughout the literature to radiation therapy [5-14] for these tumors; however, this mode of treatment seems to have met with universal failure. This is likewise true of some of the cases which we are presenting. The mode of treatment which appears to offer the most promise is radical surgery using the mastoid approach.[13] In none of our cases has there been evidence of recurrence to the present time following adequate surgery. It may be noted that these cases are relatively recent, and the possibility of recurrence in the future cannot be eliminated.

Summary

We have reported four cases of nonchromaffin paraganglioma of the middle ear, seen in this hospital during the last three years. In all cases the treatment of choice was radical surgery, and to date no recurrences have been found.

Cooperation was given by Dr. Louis E. Adin (Case 1), Dr. Lloyd F. Ritchey (Case 2), and Dr. Lyle M. Sellers (Cases 3 and 4).

3500 Gaston Ave.

REFERENCES

1. Blomqvist, E.; Lindwall, F., and de Mare, G.: Glomus Jugulare Tumors in the Ear, Acta otolaryng., Supp. 116, pp. 50-56, 1954.

2. Dockerty, M. B.; Love, J. G., and Patton, M. M.: Nonchromaffin Paraganglioma of Middle Ear: Report of Case in Which Clinical Aspects Were Those of Brain Tumor, Proc. Staff Meet. Mayo Clin. 26:25, 1951.

3. Gaffney, J. C.: Carotid-Body-like Tumours of the Jugular Bulb and Middle Ear, J. Path. & Bact. 66:157-170, 1953.

4. Graf, K.: Über die nichtchromaffinen Paragangliome des Mittelohrs, Ztschr. Laryng., Rhin. Otol. 32:619-626, 1953.

5. Hooper, R. S.: The Glomus Jugulare Tumor: Clinical and Radiological Features, J. Fac. Radiologists 7:77-89, 1955.

6. Huppler, E. G.; McBean, J. B., and Parkhill, E. M.: Chemodectoma of the Glomus Jugulare: Report of a Case with Vocal Cord Paralysis as a Presenting Finding, Proc. Staff Meet. Mayo Clin. 30:53-58, 1955.

7. Lattes, R.: Nonchromaffin Paraganglioma of Ganglion Nodosum, Carotid Body, and Aortic-Arch Bodies, Cancer 3:667-694, 1950.

8. Lattes, R., and Waltner, J. G.: Nonchromaffin Paraganglioma of the Middle Ear (Carotid-Body-like Tumor; Glomus-Jugulare Tumor) Cancer 2:447-468, 1949.

9. Leray, G.: Glomus Jugulare Tumor: 4 Cases, Ann. Otol. 71:39-66, 1954.

10. McNeill, K. A., and Millner, G. A.: Bilateral Tumors of the Glomus Jugulare, J. Laryng. & Otol. 69:430-431, 1955.

11. Rosenwasser, H.: Carotid Body Tumor of the Middle Ear and Mastoid, Arch. Otolaryng. 41:64-67, 1945.

12. Seiffert, A., Zentralbl. Hals-, Nasen- u. Ohrenh. 35:348, 1933-1934; cited by Gaffney.[3]

13. Shambaugh, G. E., Jr.: Surgical Approach for So-Called Glomus Jugulare Tumors of the Middle Ear, Laryngoscope 65:185-198, 1955.

14. Siekert, R. G.: Neurologic Manifestations of Tumors of the Glomus Jugulare: Chemodectoma, Nonchromaffin Paraganglioma or Carotid-Body-like Tumors, A. M. A. Arch. Neurol. & Psychiat. 76:1-13, 1956.

15. Strong, M. S.: Glomus Jugulare Tumor of the Middle Ear: A Clinical Note, A. M. A. Arch. Otolaryng. 60:145-148, 1954.

16. Weille, F. L., and Lane, C. S.: Surgical Problems Involved in the Removal of Glomus Jugulare Tumors, Laryngoscope 61:448-459, 1951.

17. Williams, L. H.; Child, D. S.; Parkhill, E. M., and Pugh, D. G.: Chemodectomas of the Glomus Jugulare (Nonchromaffin Paragangliomas) with Especial Reference to Their Response to Roentgen Therapy, Ann. Otol. Rhin. Laryng. 64:546-566, 1955.

18. Winship, T.; Klopp, C. T., and Jenkins, W. H.: Glomus-Jugularis Tumors, Cancer 1:441-448, 1948.

Chemodectoma of the Trachea

JULIUS W. McCALL, M.D., and FARID K. KARAM, M.D., Cleveland

Recently we had under our care at Saint Luke's Hospital a case of chemodectoma of the trachea. Because of the rarity of the condition, we felt that a presentation of this case might be of particular interest.

References to tumors of this type are quite infrequent. The existence of the chemoreceptors was first noted in 1743 by von Haller, who described a soft, ovoid, or irregular, tough, pale tan structure on the medial wall of the common carotid at its bifurcation, and gave it the name "ganglion minutum." This is the structure we now know as the carotid body. The first notation of a tumor of this structure was made by Marchand in 1891.

In the mediastinum a number of chemoreceptor bodies have been described in relation to the arch of the aorta, the ductus arteriosus, and the roots of the great vessels, the first observations being those of Biedl and Wiesel in 1902.

In 1941 Guild described a small cellular body in sections of the temporal bone which is known as the glomus jugulare.

In Burman's comprehensive article, "The Chemodectoma," he quotes several authors as having located and reported the carotid bodies near the innominate artery, either near its bifurcation into the subclavian or common carotid arteries, or lateral to the origin of the subclavian artery. Another is situated on the anterolateral aspect of the left portion of the aortic arch near the origin of the left subclavian artery. A third is found near the pulmonary end of the ductus arteriosus. An inconsistent chemoreceptor organ is located near the origin of the left coronary artery.

Accepted for publication June 24, 1957.

From the Department of Laryngology, St. Luke's Hospital.

The first case in the literature of carotid-body tumor of the trachea was recently reported by Zeman, who suggested that it originated from misplaced carotid-body tissue in the wall of the trachea. The presenting symptom of this case was sudden hemoptysis after a paroxysm of coughing. The tumor was found in the left upper portion of the trachea just below the vocal cords. The tumor was successfully removed and the area cauterized, with no recurrence after three years.

Report of Case

A 67-year-old white woman was admitted to Saint Luke's Hospital because of difficulty in breathing. One month prior to admission, the patient started having difficulty in breathing and swallowing, becoming progressively worse and associated with hoarseness for the two weeks prior to admission. This had become severer until three days prior to admission, when she had not been able to eat or sleep.

Physical examination showed a blood pressure of 140/90 and a pulse of 100. She was a well-developed woman with marked respiratory difficulty. Chest examination revealed short labored inspirations and expirations with sounds like "breathing in a tunnel." The heart was normal in rhythm and was not enlarged; the abdomen was negative. Examination of the larynx by indirect laryngoscopy revealed copious mucus. The larynx as a whole was shifted markedly to the left. The right vocal cord was white, large, irregular, and did not move on phonation, and the left vocal cord seemed normal. Several masses of grayish-tan tissue were seen to extend below the vocal cord down the trachea. In the supraclavicular area a mass was palpated which was 2×5 cm. in greatest dimensions, and was smooth, hard, and fixed to the underlying tissue. In the neck there was a firm, hard, somewhat nodular mass in the midline over the trachea; no lymph nodes were palpated in the neck. Laboratory tests were within normal limits. An x-ray of the chest revealed no evidence of pulmonary infiltration, mediastinal masses, or cardiac enlargement. There was a small left para-

Fig. 1.—Chemodectoma. Low power, taken from the trachea.

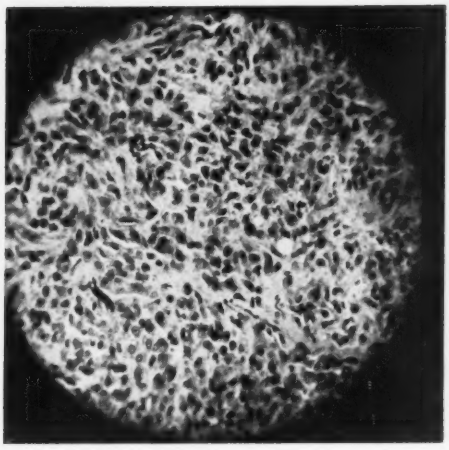


Fig. 2.—High power shows the typical epithelioid cells, which are polyhedral, with fine granular cytoplasm and large vesicular pale nuclei.

superior mediastinal calcification measuring about 1.5 cm. in greatest dimension, which was interpreted as degenerate calcification in either the cervical lymph nodes or the thyroid adenoma. Eight hours after admission the patient's respiration became more labored, and she was taken to surgery for bronchoscopy and a tracheotomy. When the bronchoscope was passed into the trachea, the tracheal tumor bled so profusely that the patient developed an acute respiratory obstruction with anoxia and shock, later followed by cardiac arrest. The tracheotomy was done immediately, and the chest was opened and cardiac massage begun.

When the trachea was opened, the tumor mass was found to extend from the larynx to the carina, almost completely obstructing the tracheal lumen. It was impossible to give the patient an adequate airway. After 10 minutes of cardiac massage with no response, she was pronounced dead.

Permission for an autopsy was refused, but several biopsy specimens were taken from the tumor. Histopathological examination of the tissue was interpreted as malignant tumor of the chemoreceptor organ.

From the known facts in this case, the origin of the tumor cannot be determined exactly. However, the history, the symptoms, the absence of mediastinal masses, and the extent of the lesion within the trachea indicate the trachea to be the most probable site of origin.

2060 E. 9th St. (15).

BIBLIOGRAPHY

Burman, S. O.: The Chemoreceptor System and Its Tumor—the Chemodectoma, Internat. Abstr. Surg. 102:330-341, 1956, in Surg. Gynec. & Obst., April, 1956.

Gilbert, J. G.; Mazzarella, L. A., and Feit, L. J.: Primary Tracheal Tumors in Infant and Adult, A. M. A. Arch. Otolaryng. 58:1-9 (July) 1953.

Guild, S. R.: A Hitherto Unrecognized Structure, the Glomus Jugularis in Man, Anat. Rec. 79 (Supp.):28, 1941.

von Haller, cited by Burman.

Marchand, cited by Burman.

Zeman, M. S.: Carotid Body Tumor of the Trachea: Glomus Jugularis Tumor, Tympanic Body Tumor, Nonchromaffin Paraganglioma, Ann. Otol. Rhin. & Laryng. 65:960-962 (Dec.) 1956.

Endoesophageal Skin Graft in Corrosive Strictures

BLAIR FEARON, M.D., F.R.C.S. (C), and A. W. FARMER, M.D., Toronto

Although strictures of the esophagus due to corrosives in recent years are relatively less frequent than in the past, owing largely to the efforts of Dr. Chevalier Jackson, nevertheless strictures due to such materials still occur.[1,2] In our department at The Hospital for Sick Children in the past five years, although we have had 19 patients with strictures of the esophagus due to corrosives, strictures due to congenital abnormalities of the esophagus have been seen more commonly. Most noteworthy in this respect is stricture following the operation for tracheoesophageal fistula with stenosis of the esophagus and that following surgery on the esophagus where an anastomosis is carried out. In this period, treatment by dilatation has been necessary for 35 patients following operation for tracheoesophageal fistula, 8 patients with stricture as a result of hiatal hernia or congenital short esophagus, and 3 patients with stricture following other surgery.

Fortunately such postoperative strictures are thin and respond very well to regular dilatation by the method of Dr. Gabriel Tucker.[3] In our experience, dilatation after the first 10 months following the operation is rarely necessary.

In strictures of the esophagus due to corrosives, the length of time necessary to carry on treatment by dilatation is notoriously long. It is seldom that an adequate lumen can be reestablished under one or two years, and frequently an occasional dilatation will be necessary over one or two years.[4] Surgical resection of the stricture is not always the answer to the problem, for strictures at the site of an anastomosis may occur which may themselves require treatment by dilatation.[5] Furthermore, the danger of reflux esophagitis following surgery for stricture of the esophagus is a very definite one.[6]

Since 1920, corrosive agents, mainly lye, were responsible for strictures that required treatment by esophageal dilatation in 82 cases that were recorded as being seen in the department of otolaryngology at The Hospital for Sick Children. There were 11 deaths in this series, and the average duration of treatment for the remainder was six years. This includes 12 patients that are under treatment or observation at the present time.

In 1947, in an attempt to better the state of affairs, one of our former staff members, Dr. J. G. Strachan, and one of us (A. W. F.) applied a skin graft at the stricture site. Since the principle is similar to that which we shall describe, details of the method used at this time will not be presented. Skin graft was then attempted on three patients. One of these was thought to be partially successful, although six years of further treatment by dilatation was required. The other two patients are still under treatment, and one of them has had as well two unsuccessful surgical repairs by resection of the stricture.

The principle for using the skin graft in the esophagus is the same as that for skin grafting of surface burns, namely, to prevent contracture. The mucosa of the esophagus resembles skin to the extent that it is covered with noncornified stratified squamous epithelium with an outer germinal layer.[7] It was considered that if a graft of skin could be successfully applied to the area of stenosis in the esophagus, that possibly the marked stricture could be avoided. Ideally it would be best to apply the graft before stricture formation could occur, that is, at about the end of two weeks. However, very few of the cases are seen in the early stages, and again it is difficult to determine which burns will result in severe strictures. If a fresh extensive lye burn of the esophagus is admitted to our service, the usual emergency treatment is carried out and the patient's hydration and nutrition maintained by intravenous therapy until normal swallowing occurs. When the swelling and edema has subsided, (after 7 to 10 days,) esophagoscopy under general anesthesia is proceeded with to determine the extent of the damage. At this stage the

Submitted for publication June 22, 1957.

Presented at the Fifth Pan-American Congress of Otorhinolaryngology and Bronchoesophagology, San Juan, Puerto Rico; April 11, 1956.

Department of Otolaryngology, University of Toronto, and The Hospital for Sick Children (Dr. Fearon); Assistant Professor of Surgery, University of Toronto; Surgeon-in-Chief, The Hospital for Sick Children; Chief Consultant, Surgery, Royal Canadian Air Force (Dr. Farmer).

ulcerated surface may still be coated with slough or may identify itself by its inflamed granular appearance. Leegaard[8] suggests that methylene blue swallowed before the procedure will distinguish the normal mucosa from the area of mucosal ulceration. We have not yet had the opportunity to try a skin graft on a recent corrosive injury of the esophagus.

To accomplish a successful skin graft in the corrosive stricture of the esophagus, we have set forth the following conditions:

1. There must be a gastrostomy and a string must be present in the esophagus and out of the mouth and of the gastrostomy.
2. An adequate lumen must be present at the site of the stricture.
3. There must be a semirigid mold on which may be applied the skin to be grafted in apposition to the lumen surface of the stricture area. This mold must be necessarily of a nonirritating substance as well as being malleable to the extent of shaping it properly to take its place in the esophagus for a considerable period of time. This mold should be of such a shape that the laryngeal or tracheal airway is not obstructed. Also it is best to have it flared at the upper end to prevent the mold from slipping downward and tapered at the distal end in order to introduce it easily into the stricture as well as to remove it easily after its use has been served.
4. Provision must be made to anchor the mold in such a way that it will not slip out of position and yet have sufficient mobility to move with esophageal movement during swallowing.
5. The surface of the stricture area must be raw in order for the skin to "take."
6. If the mold is not radiopaque, a piece of metal should be placed in some part of the tube so that its position may be checked by x-ray while the mold is in place.
7. An adequate area of split-thickness autogenous skin graft (0.012 to 0.014 in. in thickness) must be made available at the time of operation and this skin fixed firmly to the mold with the cut surface outwards.
8. The mold with the attached skin must be left in position long enough to permit the skin to "take."

When the patient does not have a gastrostomy and it has been decided to carry out a skin graft procedure, then the gastrostomy must be performed. At this time a string may be threaded through the esophagus by introducing a small bougie through the esophagoscope into the stomach, and the surgeon may tie a string on this bougie when he has the stomach open, and thus bring the string up into the mouth. Except for this, and aside from having the child swallow the string, there are other methods of stringing the esophagus that we have found valuable.

The method that has been found to be most successful and one which may be applied to most patients of any size (the smallest being 3 lb. 13 oz.) is that of employing the principle of the magnet. A 3 mm. Norris Alnico magnet is fastened to the end of a No. 8 French rubber duodenal tube. A vent is made in the side of the tube near the magnet in order to inflate the stomach. The stricture opening must be at least a No. 10 French in size to permit the magnet and tube to pass. The magnet on the tube is then passed via the esophagoscope into the stomach. (This may be passed in the same manner as a feeding tube, in many instances, without the use of the esophagoscope.) Next a long ferrous instrument is passed into the stomach through the gastrostomy. (A long curved Kelly forceps is very satisfactory for this purpose). The stomach is inflated through the tube and the magnet manipulated until it is felt to attach itself to the forceps. The latter is then carefully withdrawn from the stomach with the attached magnet and tube. The string is fastened to the tubing and brought up through the esophagus and out through the mouth. Although fluoroscopy is not essential, it serves to speed up the procedure greatly.

In the situation where the lumen of the stricture is very small and difficult to identify from above, it has been necessary to pass the bougie in a retrograde fashion, taking advantage of the funnel shape of the distal segment of the esophagus leading upward to the stricture. In small children it has been found that by introducing an esophagoscope through the gastrostomy into the stomach and inflating the stomach, the distal segment of the esophagus will also inflate, and by fluoroscopic guidance the esophagoscope is readily directed into the distal esophagus from below. A No. 5 ureteral bougie is then easily passed through the stenosis from below up into the mouth, and thus the stringing of the esophagus may be carried out.

An adequate lumen through the stricture must be acquired so that the lumen in the final result will be adequate. If the skin graft were attempted before the area of burn went on to fibrosis and stenosis, then this condition would not apply. In the two cases which subsequently will be described, quite marked stenosis had occurred, and therefore it was necessary to dilate the stricture in order to bring the lumen to a satisfactory point for skin graft. The size of the stricture lumen would depend upon the age and size of the child. In each of our cases a No. 36 French was the goal which was set. The most satisfactory method of

dilating such a stricture is that of Dr. Gabriel Tucker, namely by retrograde dilatation with the Tucker bougies.[2] To bring this stricture up to the desired size quickly the principle of indwelling bougies was employed, and in this manner the stricture size may be increased very rapidly and with very little loss of time in the course of the patient's treatment.[9] For the mold to which to attach the skin to be used for the grafting, polyethylene tubing is found to be the most suitable material available for this particular purpose, because it is nonirritating, light, quite malleable when heated to the proper temperature, and keeps its shape at body temperature. It is also readily cut with scissors or knife to the particular shape indicated for the patient.

In the two case histories being reported the stricture in each was in the upper third of the esophagus, which meant that a cylindrical tube would press on the upper trachea, probably causing laryngeal and/or tracheal obstruction. This complication was avoided by flattening the proximal one-third of the tube, and in neither case was there respiratory distress from the airway being obstructed at any time during the presence of the polyethylene mold in the esophagus. The edge of the proximal end of the tube was slightly flanged to help hold the mold in place. The lower end was tapered to permit the mold to be introduced into the stricture easily and also to permit its easy removal through the gastrostomy when the mold was no longer necessary.

To hold the mold in place from above, a dentist fixed a band with a wire loop to a lower second molar tooth. To this loop was attached the string leading from the proximal end of the tube. A second string was attached to the tip of the distal end of the tube and out through the gastrostomy, tied to a large wooden button, and taped to the anterior abdominal wall. Enough slack in the string was provided to enable some movement of the mold with the normal movement of the esophagus in swallowing (Fig. 1).

In order for the skin graft to take in the area of the fibrosis which is causing the stricture, it is necessary to provide a raw surface to which to apply the skin, and in doing so remove a certain depth of the fibrous tissue. This is accomplished in the following manner. A sanding instrument is prepared by cementing a coarse waterproof sandpaper closely and firmly to the dilating portion of a No. 32 Jackson esophageal bougie. The stricture site is exposed through a large esophageal speculum, and the sanding instrument is introduced through the speculum to the area of the scar. The surface over the scar is abraded with a to-and-fro movement until the area is completely denuded and freely bleeding. It is difficult to estimate how deep this area must be or actually how deep one has gone with the abrasion. However, all the area of abnormal epithelium may be removed in this manner, leaving a raw bleeding surface. Particular attention must be paid to the lower end of the stricture area for this is quite easily missed in this process.

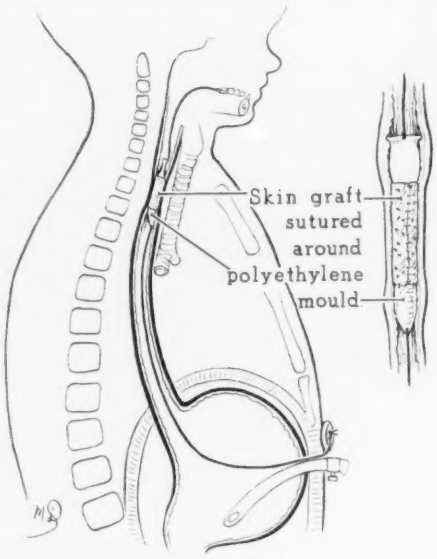


Fig. 1.—Diagram showing mold with attached skin graft in position in the area of the cicatricial stricture of the esophagus.

While the esophagus is being prepared to receive the skin graft, enough skin is removed from the donor site to be more than sufficient to cover the area in the esophagus to be grafted. This is 0.012 or 0.014 in. in thickness. The skin is then sutured to the mold with the cut surface out, many fine silk sutures being used so that the polyethylene mold is enclosed in the tube of skin. A circle of stainless steel wire is sutured in the tube to provide an identifying point in order to check the position of the skin graft from time to time by x-ray.

The polyethylene mold covered by the skin is then guided into the esophagus by traction on the string attached to the lower end of the tube and leading out through the gastrostomy. This is best accomplished by using a No. 38 Tucker indwelling retrograde bougie to immediately precede the mold with the graft, thus helping to reduce trauma to the graft while it is passing through the stricture. When the proximal end of the mold is in the esophagus, its position is checked with the esophagoscope, and the tube graft is further advanced until it is in proper position within the stricture. At this point the polyethylene mold is anchored at both ends as previously described. This stage

of the operation is now completed and the mold with the skin is left in position for one month. In this interval feeding is carried out essentially by the gastrostomy tube, although we permit the patient to have clear fluids by mouth after the first 48 hours or so. At the end of four weeks the mold is removed by drawing the tube downward into the stomach and out through the gastrostomy by traction on the string.

It is preferred not to carry out esophagoscopy until the end of another month or so, when any reaction within the esophagus should have subsided. Esophagoscopy at this time readily reveals the site of the graft, if it has taken, as a rather pearly gray color in contrast to the normal esophageal mucosa.

We shall now present two cases of corrosive stricture of the esophagus in which skin graft was used as the method of treatment.

CASE 1.—A boy 2 years and 8 months old was admitted to The Hospital for Sick Children on April 17, 1953. The child had been in good health until the day of admission, when early in the afternoon, while playing in a neighbor's garage, he swallowed cleaning solution containing lye. He was immediately given the yolk of an egg by his parents and this made him vomit, and he continued to vomit until the time of admission to hospital. Examination revealed a normally developed child for his age with burns of the mouth, palate, and pharynx. Otherwise the child was found to be normal. During the first five days in hospital the child was unable to swallow even liquids, and it was necessary to maintain his hydration by intravenous fluids. By the sixth day he began to swallow fluids and was able to take a soft diet without any difficulty. On April 28, 1953, 11 days after the lye burn, esophagoscopy under general anesthesia was carried out. This revealed a 6 cm. tubular area of granulation beginning about 3 cm. below the cricopharyngeus. A linear area of granulation continued from the tubular area above, down the posterior wall to approximately the mid-esophagus. On May 15, 18 days after the accident, again esophagoscopy was carried out, and at this time the particular area of granulation was found to be constricting and this was dilated directly to a No. 24 French. One week later esophagoscopy showed progress in the stenosis to about a No. 17 French in size, and again the stricture was dilated to a No. 24 French. Because of the rapid progress in the stenosis it was decided to have a gastrostomy carried out so that the safer and more efficient dilatation by the retrograde method, as described by Dr. Gabriel Tucker, could be performed.

The gastrostomy was performed on May 15, and at the same time a string was brought up through the esophagus for future treatment. Eleven days later and thirty-nine days after the lye burn, examination of the esophagus revealed the stricture to be pinpoint (Fig. 2). Dilatation was carried out every two or three days so that a good lumen was achieved, but if the dilatations were allowed to be left longer than a week, the stenosis would become more difficult to dilate. Because of this fact, and because the patient lived some distance away from the city, and thus away from treatment, it was decided that a skin graft to the esophageal stricture should be attempted.

Fig. 2.—Barium swallow showing the cicatricial stricture of the esophagus prior to the skin-graft operation.

Preparations for the skin grafting were made as outlined earlier in the paper. The stenosis was brought up to a No. 36 French by the use of an indwelling tube. Since this stricture was high in the esophagus, it was necessary to flatten the proximal half of the polyethylene tube (No. 34 French) being used to hold the skin in place in the stricture. The skin graft operation was carried out uneventfully on Aug. 31, 1953. At no time did the patient have any discomfort or reaction while the polyethylene tubing with the skin was in place. One month after the operation the polyethylene mold was removed easily and brought out through the gastrostomy, leaving the skin behind in the esophagus. One month after the removal of the tube the esophagus was reexamined. We were very fortunate at this time to have Dr. Gabriel Tucker present for his opinion and comment. A 7 by 35 full-lumen esophagoscope was introduced into the esophagus, and the area of skin graft easily identified because of the pearly-gray color. There seemed to be some narrowing at the lower end of the graft, although

Fig. 3.—Barium swallow four years after lye burn.

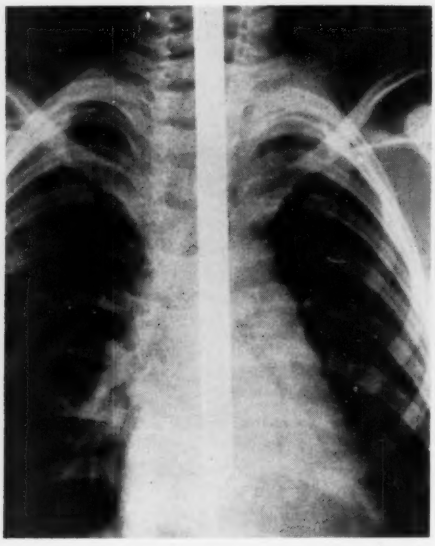
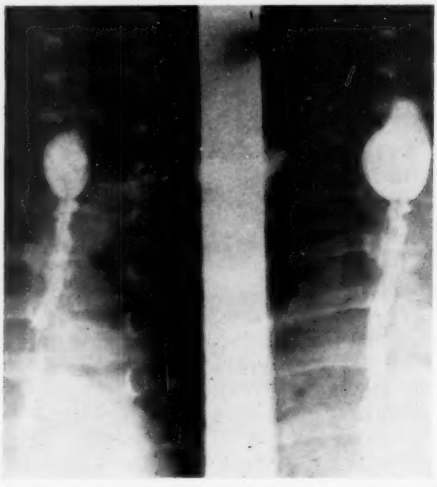


Fig. 4.—X-ray showing a 6 mm. Lell esophagoscope in esophagus well beyond the site of the skin graft four years after operation.

Dr. Tucker felt that this was more apparent than real. The same esophagoscope passed easily through the graft into the stomach without any evidence of trauma. The gastrostomy was allowed to close, and the child was discharged Nov. 8, 1953, again swallowing normally. The esophagus has been reexamined periodically since then, and to date there has been no appreciable narrowing of the lumen at the site of the corrosive injury. The esophageal lumen at the site of the stricture is not truly a normal one, since there is a web-like narrowing where the lower end of the skin graft was judged to have been. It would appear that the skin graft had taken over most of the length of the stenosis though leaving a narrow band of scar at this one point, but nevertheless the lumen does admit a No. 7 full-lumen esophagoscope. In view of the fact that the child has been in excellent health and no active treatment has been necessary for three and a half years, it is considered that in this patient an endoesophageal skin graft was a decided success, particularly when the total treatment time was only four and a half months (Figs. 3 and 4).

Case 2.—This 4-year-old boy was admitted to The Hospital for Sick Children on June 28, 1953, after treatment for a period of a year in another hospital for a corrosive stricture of the esophagus. In March, 1952, at approximately the age of 3 years, he had swallowed a 50% lye solution. On admission to hospital he was very malnourished and dehydrated, unable to swallow even water at the time of admission. Esophagoscopy on July 3, 1953 revealed a marked stenosis at the junction of the upper third and middle third of the esophagus (Figs. 5 and 6). Gastrostomy was then performed, and at the same time a string was passed through the esophagus. The stricture responded well to retrograde dilatation, and the boy was trained to swallow a Hurst mercury bougie and on discharge from hospital was able to swallow a No. 30 French mercury bougie at

Fig. 5.—Barium swallow showing the cicatricial stricture of the esophagus prior to skin grafting.



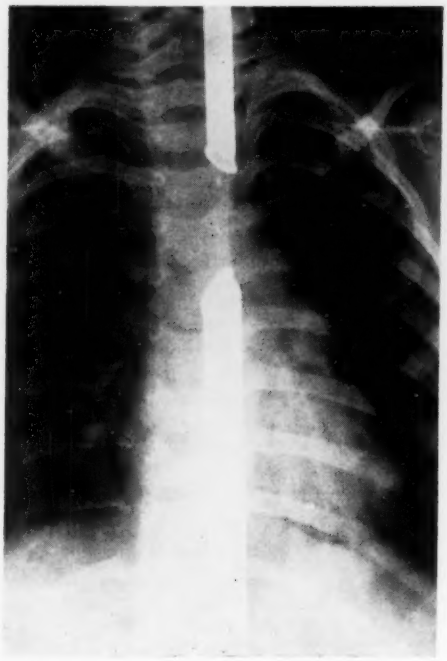


Fig. 6.—Simultaneous direct and retrograde esophagoscopy to indicate the length of the stricture.

Fig. 7.—Barium swallow three years after the skin graft operation. (The arrows indicate the site of the original stricture.)

will. A few weeks after discharge from hospital he was readmitted, on Sept. 16, 1953, with paralytic poliomyelitis involving both legs. Treatment to the esophagus had to be postponed at this stage, and when it was restarted again the stenosis was quite marked. In view of the success with the skin graft on the first patient, the same procedure was carried out on this child on Feb. 9, 1954. At this time the stenosis was forcefully dilated to a No. 40 French and the skin graft easily inserted as previously described. However, in the recovery room after the operation, the child showed marked subcutaneous emphysema and later proved by x-ray to have a mediastinal emphysema as well. The postoperative course was a stormy one, including a mediastinal abscess which, largely by good fortune, drained into the esophagus. Subsequent esophagoscopy showed a thin web and an angulation of the esophagus where the esophagus was thought to have been perforated at the time of the skin graft procedure. This necessitated subsequent retrograde dilatation and later self-treatment using the Hurst mercury bougie. A recent barium swallow showed a marked improvement in the status of the stricture over the original x-ray, and esophagoscopy revealed a narrowing and angulation as previously noted, but permitted the passage of a No. 6 by 45 full-lumen esophagoscope without evidence of trauma to the esophagus (Fig. 7).

Although the result in skin grafting the esophagus in this particular patient has been far from perfect, nevertheless a certain degree of success was achieved. No active treatment has been necessary to the stricture of the esophagus for about one and a half years. Incidentally, the patient is receiving treatment periodically to correct the deformities to his legs which resulted from the poliomyelitis. In the future we would not attempt to split the stenosis as we did in this second, patient, but rather rely on operating on the areas of scar in the esophagus to acquire a proper site on which to place the skin graft.

Summary and Conclusion

Although the proportion of strictures of the esophagus due to congenital abnormalities and their sequelae is becoming much greater owing to advances in surgery in recent years, strictures of the esophagus due to corrosive injuries still occur, and the basic management of such strictures has shown no great change. In this paper there has been reported the technique of using a skin graft to cover the area of scarring in the esophagus

due to a corrosive injury in the hope of preventing or curing a severe stricture of the esophagus, and therefore reducing the length of time of treatment required for such patients. Two case histories have been presented in which a skin graft has been used to treat such a stricture, one of these being successful, and the other only partially so. Lack of complete success in the latter can probably be attributed to faulty technique which could have been avoided. Even if the skin graft were to be a complete failure, it does not prevent the institution of other forms of treatment.

170 S. George St. (5).

REFERENCES

1. Jackson, C.: Esophageal Stenosis Following the Swallowing of Caustic Alkalies, J. A. M. A. 77:22, 1921.

2. Jackson, C. and Jackson, C. L.: Broncho-esophagology, Philadelphia, W. B. Saunders Company, 1950, p. 306.

3. Tucker, G.: Cicatricial Stenosis of the Oesophagus with Particular Reference to Treatment by Continuous String Retrograde Bouginage with the Author's Bougie, Ann. Otol., Rhin. Laryng. 33:1180, 1924.

4. Woodward, F. D., in Otolaryngology, edited by G. M. Coates, H. P. Schenck, and M. V. Miller, Hagerstown, Md., W. F. Prior Company, Inc., Vol. 5, Chap. 11, p. 23.

5. Cody, C. C., III.: Recent Trends in the Management of Esophageal Strictures, Ann. Otol. Rhin. & Laryng. 63:1120-1139, 1954.

6. Editorial: The Importance of the Esophago-Gastric Valve Mechanism in Surgery of the Stomach and Esophagus, Surg., Gynec., & Obst. 94:502, 1952.

7. Scott-Brown, W. G., Editor: Diseases of the Ear, Nose and Throat, New York, Paul B. Hoeber, Inc. (Medical Book Department of Harper & Brothers) Vol. 2, p. 383.

8. Leegaard, T.: Corrosive Injuries of the Oesophagus, with Particular Reference to the Treatment of Acute Oesophagitis, J. Laryng. & Otol. 60:389, 1945.

9. Tucker, G.; Lautz, V.; Tracy, J. B., and Fearon, B. W.: The Diagnosis and Treatment of Benign Stenosis of the Esophagus, with Special Reference to Treatment by Dilatation with the Indwelling Cannulated Bougies, Ann. Otol. Rhino. & Laryng. 59:823-836, 1950.

News and Comment

ANNOUNCEMENTS

Examination by American Board of Otolaryngology.—The American Board of Otolaryngology will conduct only one examination in 1958, and this will be Oct. 6-9, 1958, in Chicago, at the Palmer House.

The American Academy of Ophthalmology and Otolaryngology, through its Subcommittee on Hearing in Children of the Committee on Conservation of Hearing, has been conducting a long-term nation-wide study of problems relating to the conservation of hearing in children. The specific aims are to develop the most efficient case-finding methods and to use these methods in estimating the magnitude of the problem in the country; to study state laws and review current practices and facilities for rehabilitation of hearing-impaired children; to help develop methods for medical and surgical rehabilitation standards; and ultimately to use the Subcommittee findings in assisting professional workers to improve and enhance programs in hearing loss.

In the second year of operations, a full-time Executive Director has been engaged, and offices have been established at the Graduate School of Public Health, University of Pittsburgh. An initial study is being conducted in Pittsburgh to identify early medical signs and symptoms which may indicate danger of hearing impairment, to measure the psychological, social, and other effects of such impairment and to develop efficient and economical methods for the testing of hearing in children. The Pittsburgh study is a cooperative effort among the following: The Subcommittee on Hearing in Children, the Graduate School of Public Health and the School of Medicine of the University of Pittsburgh, the Pittsburgh Board of Public Education, and the Allegheny County Department of Health.

The members of the Subcommittee on Hearing in Children are as follows: Dr. John E. Bordley, Baltimore; Dr. Victor Goodhill, Los Angeles; Dr. Hollie E. McHugh, Montreal; Dr. S. Richard Silverman, St. Louis; and Dr. Raymond E. Jordan (Chairman), Pittsburgh. An advisory committee of consultants from the University of Pittsburgh includes Dr. Samuel M. Wishik, Dr. Leo G. Doerfler, and Dr. Isidore Altman. Grants from the United States Children's Bureau through the Pennsylvania Department of Health and from the National Institutes of Health are providing financial support.

GENERAL NEWS

Specialists Certified by the American Board of Otolaryngology.—The following candidwere certified as specialists by the American Board of Otolaryngology, Oct. 7-11, 1957:

Ainslie, Robert B., Madison, Wis.
Anthony, James M., Dallas, Texas.
Ashcraft, Elijah J., Jr., Harlingen, Texas.
Belzile, Marcel J., Montreal, Canada.
Blatt, Irving M., Ann Arbor, Mich.
Bunker, Rex James, Santurce, Puerto Rico.
Carsel, Jack, Canoga Park, Calif.
Culbertson, Marvin C., Jr., Dallas, Texas.
Dale, David O., Park Ridge, Ill.
Davis, Herbert A., Allen Park, Mich.
Deatsch, Wayne W., San Francisco.
Donohoo, Albert C., San Antonio, Texas.
Edwards, Larry E., Fairfield, Conn.
Elliott, Dean C., Petoskey, Mich.
Fanjoy, Roy W., St. John, N. B., Canada.
Fischer, W. George, Stockton, Calif.
Freeman, Richard J., Lakewood, Ohio.
Goodwin, Maurice R., Bronx, New York.
Hara, Kinge, Milwaukee.
Hendrick, Robert S., Shreveport, La.
Jackson, Thomas M., Memphis, Tenn.
Jeantet, Colette, Chicago.
Joseph, Donald J., Denver.
Kaplan, Gerald, Hackensack, N. J.
Karutz, Wallace S., Brooklyn.
Kavanagh, Thomas W., Phoenix, Ariz.
Kenter, Ali Mahmut, Boston.
Kinkella, Albert M., Springfield, Ill.

Klotz, Robert E., Boston.
Knight, Robert E., Bloomington, Ill.
Kowal, Roland A., Berwyn, Ill.
Kuhn, Arthur J., Hammond, Ind.
Lilly, F. Vivan, Beckley, W. Va.
Lineback, Carl M., Fort Smith, Ark.
Lowry, Dick M., Oklahoma City.
Lubrano, Daniel, Tampa, Fla.
Maier, Godfrey George, Louisville, Ky.
Marhefka, Joseph C., Jr., Easton, Pa.
Montgomery, Amzi D., Lafayette, La.
Normann, Frank S., Jr., New Orleans.
Pennington, Claude Lee, Macon, Ga.
Rafiee, Ali Akbar, Perth Amboy, N. J.
Sade, Jacob, Boston.
Schmidt, Ruth B., Pomona, Calif.
Schosheim, Arnold M., Brooklyn.
Schultz, Marvin H., Marquette, Mich.
Shankar, Ramaiyer Gouri, Boston.
Shepard, Marvin G., Dallas, Texas.
Shramek, Joseph M., Long Beach, Calif.
Siedentop, Karl H., Oak Park, Ill.
Sorokanich, Vera, Scranton, Pa.
Spillane, James H., Phillipsburg, N. J.
Stock, Maurice F., Pittsburg, Kan.
Summerlin, Jack D., Indianapolis.
Sweeney, Francis J., Bethesda, Md.
Talmage, Walter R., Morristown, N. J.
Tippin, Ernest E., Jr., Wichita, Kan.
Trunzo, Thomas H., Latrobe, Pa.
Updegrove, Robert A., Williamsport, Pa.
Utrata, Joseph, Chillicothe, Ohio.
Vicens, Enrique A., Ponce, Puerto Rico.
Waldmann, Robert P., Seattle.
Wang, Hsin Fen, Oakland, Calif.
Warren, Claude M., Jr., Mobile, Ala.
Wolferman, Adolph, Cumberland, Md.

IF YOU CHANGE ADDRESS

Please notify us at least six weeks before you make the change.

With your notification, include the address label clipped from the latest copy of the journal. If your city has postal zone numbers, be sure to include the zone number in your new address.

You will want to get your copies promptly; so notify us at least six weeks in advance. Send your change notice to

A. M. A. Archives of Otolaryngology
American Medical Association
535 North Dearborn St.
Chicago 10, Illinois

Books

BOOK REVIEWS

Magnetic Removal of Foreign Bodies. By Murdock Equen, M.D.F.A.C.S. Price, $4.50. Pp. 94, with 119 illustrations. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill., 1957.

This monograph deals with the authors' modification of the Alnico magnet for the removal of foreign bodies in the air passages, esophagus, stomach, and duodenum.

The author presents the history of the development of the magnet, its description, method of usage, and his personal modifications. The third chapter, and the succeeding three chapters are devoted to the use of this magnet in the removal of foreign bodies from the air passages, esophagus, stomach, and duodenum. Actual case histories are presented, and these are amplified by both illustrations and roentgenograms.

The final chapter deals with the failures and their causes.

This monograph is a useful book for the bronchologist and the gastroenterologist who performs his own gastroscopies.

Diseases of the Nose and Throat. Sixth Edition. By Sir St. Clair Thomson and Sir Victor E. Negus (Revised by Sir V. E. Negus, assisted by G. H. Bateman). Price, $15.50. Pp. 1040 with 399 illustrations, including 13 color plates. Cassell & Co., Ltd., 37-38 St. Andrew's Hill, Queen Victoria St., London E. C. 4, 1955. (Orders from the United States and Canada will be filled by Cassell & Co., Ltd., Kingswood House, 1068 Broadview Ave., Toronto 6.)

This sixth edition appearing seven years after the previous edition, is timely and necessary. Many chapters have been rewritten and revised. A great deal of obsolete material has been eliminated. The general plan still follows the original format. This combines minute detail with a delightful style that makes this book one of the most popular in the field of nose and throat. Each subject is discussed in great detail and wherever indicated, is amplified by suitable illustrations and color plates.

The first half of the book deals with problems of the nose and pharynx. Included are the common and the unusual pathologic processes. The second portion is devoted to the larynx. Many chapters have been completely rewritten and brought up to date. The description of many operations has been revised in view of the newer concepts and approaches. This revision also applies to medical treatment since there is so much change in the indications and uses of the antibiotics.

Finally, emphasis is placed on many systemic conditions that may present local manifestations in the upper air passages.

In addition to an excellent formulary containing 37 prescriptions there is an extensive and detailed index. This book cannot be recommended too highly because of the wealth of information that is presented in so delightful a manner. The general practitioner and the rhinolaryngologist will find this book most valuable.

Diseases of the External Ear. By Ben H. Senturia, M.D., with the assistance of Carl F. Gessert, Ph.D.; Morris D. Marcus, M.D.; Bernard C. Adler, M.D.; Fritz M. Liebmann, M.D.; Lawrence H. Sophian, M.D.; Charles D. Carr, M.T., and Elizabeth S. Baumann, A.B. Price, $8.50. Pp. 211, with two colored plates, plus charts, tables and other illustrations. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill., 1957.

It is a truism often heard that medicine has now reached a high degree of specialization. But how high is evidenced only by taking up a book like Senturia's, in which we find some 340 names of authors who have written articles dealing with the external ear. A cursory look at the "subject index" discloses some 420 items, from Abscesses to Zinc Stearate! The gamut runs from injuries, through tumors, diseases, and malformations. In other days we used to be satisfied with a diagnosis of a wet ear, a dry ear or cerumen. Eczema covered a multitude of

conditions which were not eczematous at all. Furunculosis and external otitis were sufficient descriptive conditions. . . . Senturia and his associates have worked in the biology, chemistry, and endocrinology fields in tracking down "causes," and they admit that all may not as yet have been determined. We used to think that fungi had a great area of causation. Now we are told that "fungi have been relegated to a minor role." The Pseudomonas and Gram-negative organisms in general are prevalent in infections. All treatment must be based on a painstaking, thorough diagnosis. Hit-or-miss therapy will seldom give good results. . . . Diseases of the external ear are often difficult to cure; sometimes baffling. There is a tendency to recurrence, which may be highly disappointing. Patients suffer tortures from itching, and too often create complications by scratching. . . . After diagnosis has been well established, one can hope for relief, but "cure" comes not so often as one could wish. . . . This is an intensive and important study of a common human ailment.

Anesthesia and Otolaryngology. By Donald F. Proctor. Price, $7.00. Pp. 267, with 33 illustrations. Williams & Wilkins, Royal and Guilford Aves., Baltimore, 1957.

This book presents the problems of anesthesia from the standpoint of the physician-anesthesiologist and the otolaryngologist. The first section deals with general considerations, such as preoperative preparation of the patient, the various drugs in common usage, post-anesthetic care and some medicolegal considerations.

The second section outlines the various methods of general anesthesia for most otolaryngological procedures. Preoperative sedation is advised, but the narcotics are to be avoided. Intubation is usually the method of choice, but the dangers and the complications are presented in detail. Other techniques that are of a controversial nature are also discussed.

The third section is concerned with local anesthesia. This includes the choice of drugs, their action and possible toxic reactions, and technique for use. This includes methods of nerve block, superior laryngeal nerve block, and anesthesia for peroral endoscopy and bronchography.

The last section deals with respiratory resuscitation, obstruction of the upper airways, with indications for tracheotomy, ventilation problems in the newborn, etc.

This book is valuable for both the anesthesiologist and the laryngologist and should be of great interest and helpfulness to both.

BOOKS RECEIVED

* **The Development and Disorders of Speech in Childhood.** By Muriel E. Morley, B.Sc., F.C.S.T. Price, $9.00. Pp. 440, with many charts, tables, and illustrations. E. & S. Livingstone, Ltd., 16 and 17 Teviot Place, Edinburgh; Williams & Wilkins Company, Mount Royal and Guilford Aves., Baltimore 2, 1957.

The Glaucomas. Second Edition. By H. Saul Sugar, M.D., F.A.C.S. Price, $13.50. Pp. 516, with 164 illustrations. Paul B. Hoeber, Inc., (Medical Book Department of Harper & Brothers), 49 E. 33rd St., New York 16, 1957.

* **Ear, Nose and Throat Dysfunctions Due to Deficiencies and Imbalances.** By Sam E. Roberts, M.D. Price, $8.50. Pp. 323, with 58 illustrations. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill. (published simultaneously in the British Commonwealth by Blackwell Scientific Publications, Ltd., Oxford, England, and in Canada by The Ryerson Press, Toronto), 1957.

* To be reviewed.

Directory of Otolaryngological Societies *

FOREIGN AND INTERNATIONAL

Canadian Otolaryngological Society
(Société Canadienne d'Otolaryngologie)

President: Dr. Robert T. Hayes, 42 Cobourg St., St. John, N. B.
Secretary: Dr. Donald M. MacRae, 34 Spring Garden Rd., Halifax, N. S.
Place: Nova Scotian Hotel, Halifax, N. S. Time: June 9-11, 1958.

Venezuela Otorhinolaryngology Society

President: Dr. Alfredo Celis Pérez.
Secretary: Dr. Jesús Miralles.

International Bronchoesophagological Society

President: Dr. Jo Ono, Tokyo.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.
Meeting: Seventh International Congress of Bronchoesophagology.
Place: Kyoto, Japan. Time: Sept. 12-14, 1958.

International College of Surgeons, Section of Ophthalmology and Otolaryngology

President: Dr. E. G. Gill, 711 S. Jefferson St., Roanoke, Va.
Secretary-Treasurer: Dr. Louis Savitt, 3402 W. Lawrence Ave., Chicago 25.

International Congress of Audiology

General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.
Place: Leyden, Netherlands.

International Course in Paedo-Audiology

General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.
Place: Groningen, Netherlands.

Pan-American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

President: Dr. José Gros, Havana, Cuba.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.
Meeting: Sixth Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Place: Brazil. Time: Aug. 12, 1958.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Alden Miller, 500 S. Lucas Ave., Los Angeles.
Secretary: Dr. Hugh A. Kuhn, 112 Rimbach St., Hammond, Ind.
Place: San Francisco. Time: June 23-27, 1958.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Erling W. Hansen, 90 S. Ninth St., Minneapolis.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st Ave. Bldg., Rochester, Minn.

American Board of Otolaryngology

President: Dr. Gordon D. Hoople, 1100 E. Genesee St., Syracuse 10, N. Y.
Secretary-Treasurer: Dr. Dean M. Lierle, University Hospitals, Iowa City.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

American Broncho-Esophagological Association

President: Dr. Walter Hoover, 605 Commonwealth Building, Boston.
Secretary: Dr. F. Johnson Putney, 1719 Rittenhouse Sq., Philadelphia 3.
Place: Mark Hopkins Hotel, San Francisco. Time: May 21-23, 1958.
Place: The Homestead, Hot Springs, Va. Time: March, 1959.

American Laryngological Association

President: Dr. Harry P. Schenk, 326 S. 19th St., Philadelphia 3.
Secretary: Dr. James H. Maxwell, University Hospital, Ann Arbor, Mich.
Place: Fairmont Hotel, San Francisco. Time: May 19-20, 1958.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Lawrence R. Boies, University Hospital, Minneapolis 14.
Secretary: Dr. C. Stewart Nash, 700 Medical Arts Bldg., Rochester 7, N. Y.
Place: Mark Hopkins Hotel, San Francisco. Time: May 21-23, 1958.
Place: The Homestead, Hot Springs, Va. Time: March, 1959.

SECTIONS:

Eastern.—Chairman: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.
Southern.—Chairman: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis, Tenn.
Middle.—Chairman: Dr. Francis L. Lederer, 860 N. Lake Shore Drive, Chicago.
Western.—Chairman: Dr. Ben. R. Dysart, 960 E. Green St., Pasadena 1, Calif.

American Rhinologic Society

President: Dr. Russell I. Williams, 408 Hynds Bldg., Cheyenne, Wyo.
Secretary: Dr. Robert M. Hansen, 1735 N. Wheeler Ave., Portland, Ore.
Annual Clinical Session: Illinois Masonic Hospital, Chicago, October, 1958.
Annual Meeting: Chicago, October, 1958.

American Otological Society, Inc.

President: Dr. Dean M. Lierle, State University of Iowa, Iowa City.
Secretary: Dr. Lawrence R. Boies, University Hospitals, Minneapolis 14.
Place: Fairmont Hotel, San Francisco. Time: May 17-18, 1958.
Place: The Homestead, Hot Springs, Va. Time: 1959.

American Otorhinologic Society for Plastic Surgery, Inc.

President: Dr. Armand L. Caron, 36 Pleasant St., Worcester, Mass.
Secretary: Dr. Louis Joel Feit, 66 Park Ave., New York 16.

American Society of Facial Plastic Surgery

President: Dr. Irvin J. Fine, 505 New Brunswick Ave., Perth Amboy, N. J.
Secretary: Dr. Samuel M. Bloom, 123 E. 83rd St., New York 28.
Annual Spring Meeting: Columbus, Ohio, March 10-11, 1958.

American Society of Ophthalmologic and Otolaryngologic Allergy

President: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis 3.
Secretary-Treasurer: Dr. Michael H. Barone, 468 Delaware Ave., Buffalo 2.

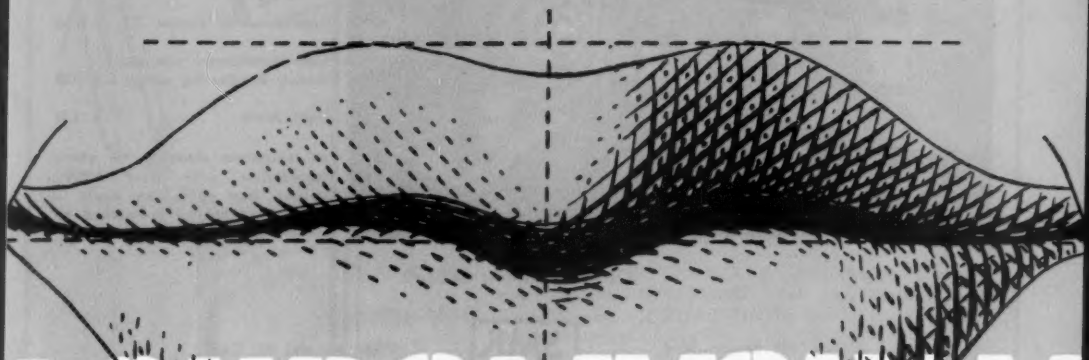
Otosclerosis Study Group

President: Dr. Joseph A. Sullivan, 174 St. George St., Toronto 5, Canada.
Secretary-Treasurer: Dr. Arthur L. Juers, 611 Brown Building, Louisville.

Society of Military Otolaryngologists

President: Capt. William C. Livingood, U. S. N. (MC).
Secretary-Treasurer: Lt. Col. Stanley H. Bear, USAF(MC), 3810th USAF Hospital, Maxwell AFB, Alabama.

TASTY, FAST-ACTING ORAL FORM OF CITRATE-BUFFERED ACHROMYCIN V



ACHROMYCIN V*

TETRACYCLINE BUFFERED WITH SODIUM CITRATE

SYRUP

aqueous

ready-to-use

freely miscible

- accelerated absorption in the gastro-intestinal tract
- early, high peaks of concentration in body tissue and fluid
- quick control of a wide variety of infections
- unsurpassed, true broad-spectrum action
- minimal side effects
- well-tolerated by patients of all ages

ACHROMYCIN V SYRUP:
Orange Flavor. Each teaspoonful (5 cc.) contains 125 mg. of tetracycline, HCl equivalent, citrate-buffered. Bottles of 2 and 16 fl. oz.

DOSAGE:
6-7 mg. per lb. of body weight per day.

*Reg. U. S. Pat. Off.

Lederle

LEDERLE LABORATORIES DIVISION
AMERICAN CYANAMID COMPANY
PEARL RIVER, NEW YORK

Chair—Upholstered, reclining ..$150.00

Cabinet—Stainless steel, eight drawers with or without complete transilluminator, cautery rheostat and transformer, waste container, air regulator, gauge, tubing and cutoff ..$115.00 to $195.00

Light—Telescopic .....$ 16.50

Cuspidor—With Suction .....$ 95.00

Stool—Upholstered, with easy running ball-bearing casters ..$ 20.00

Light Shield .....$ 5.00

All equipment shown in the above reproduction (with spray bottles, stainless steel cotton jars, ready to be attached to your compressed air system, $531.50 F.O.B. Factory.

Catalogue sent on request

1901-1905 Beverly Blvd. SMR Los Angeles 57, Calif.

GILL MEMORIAL EYE, EAR AND THROAT HOSPITAL

Announces to the Profession

THIRTY-FIRST ANNUAL SPRING CONGRESS

in

OPHTHALMOLOGY AND OTOLARYNGOLOGY

April 14 through April 19, 1958

GUEST SPEAKERS

David B. Allman, M.D.	Atlantic City, New Jersey
Edwin N. Broyles, M.D.	Baltimore, Maryland
John J. Conley, M.D.	New York, New York
George Crile, Jr., M.D.	Cleveland, Ohio
Fred W. Dixon, M.D.	Cleveland, Ohio
Leon Goldman, M.D.	Cincinnati, Ohio
Roscoe J. Kennedy, M.D.	Cleveland, Ohio
Perrin H. Long, M.D.	Brooklyn, New York
Donald J. Lyle, M.D.	Cincinnati, Ohio
Sylvester C. Missal, M.D.	Cleveland, Ohio
C. Stewart Nash, M.D.	Rochester, New York
Edward W. D. Norton, M.D.	New York, New York
W. E. Pembleton, M.D.	Richmond, Virginia
Winston H. Price, Ph.D.	Baltimore, Maryland
Donald M. Shafer, M.D.	New York, New York
Benjamin H. Shuster, M.D.	Philadelphia, Pennsylvania
Byron Smith, M.D.	New York, New York
Norah duV. Tapley, M.D.	New York, New York
Richard C. Troutman, M.D.	Brooklyn, New York
Henry P. Wagener, M.D.	Rochester, Minnesota
James W. Watts, M.D.	Washington, D. C.
Lorenz E. Zimmerman, M.D.	Washington, D. C.

For Further Information Write:

Superintendent, P. O. Box 1789, Roanoke, Virginia

FROM OUR CATALOG NO. 18
1957 ISSUE

Cat. No. 731/S RISH OSTEOTOME, (Original Model) stainless steel, overall length 7 inches (18 cm), $9.50 each. Designed specifically for intranasal lateral osteotomy.

Features:

Only one instrument required for both sides.—A short, blunt, nonbreakable guard prevents splintering of the frontal bone. A guide, which can be felt manually through the skin, follows the exact course of the instrument.—A collar (ledge) over the entire dorsal aspect of the instrument, which when pressure is exerted, lets the osteotome stay on its desired course, and will keep it from slipping medially, causing damage to intranasal structures or laterally affecting the skin. Width of 6 mm. is provided to avoid injury to intranasal structures. Uniform thickness of 1 mm. extending for 1½ inches (38 mm) from cutting edge to prevent separation of nasal bone fragments with premature low fracture.

Ref. Archives of Otolaryngology, February 1953

B. J. FLORSHEIM

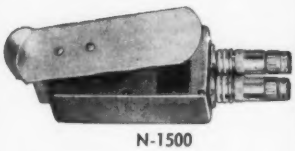
2067 Broadway, at 72nd St., New York 23, N. Y.

storz
Surgical Instruments

READY FOR DELIVERY

EXPLOSION-PROOF

JORDAN-DAY
Bone Engine



N-1500

N-1500 Bone Engine, JORDAN-DAY: explosion proof, Underwriter Laboratories listed. Complete with triple-arm, stand and explosion-proof foot control rheostat, but without handpiece. The engine and arm assembly can be sterilized by autoclaving. Motor is sealed against moisture. In addition this engine has the highest speed of any unit available $363.75

N-1502 Bone Engine, JORDAN-DAY: explosion-proof engine and foot control rheostat only $287.50

N-1504 Bone Engine, JORDAN-DAY: standard model, not explosion proof. Complete with triple-arm, stand and foot control rheostat, but without handpiece $252.25

N-1506 Bone Engine, JORDAN-DAY: standard model engine and foot control rheostat only $177.50

N-1510 Rheostat, Foot Control: to fit N-1504. The same footswitch as furnished with No. N-1500 explosion-proof unit, but can be adapted to No. N-1504 standard JORDAN-DAY engine $98.50

N-1520* Handpiece, CHAYES: No. 3, with double pulley for selection of speed. Recommended for use with the JORDAN-DAY engine. Bearing system reduces heat caused by high speed. Special large locking wheel to prevent loosening of burrs. Can be autoclaved. Stainless Steel $78.75

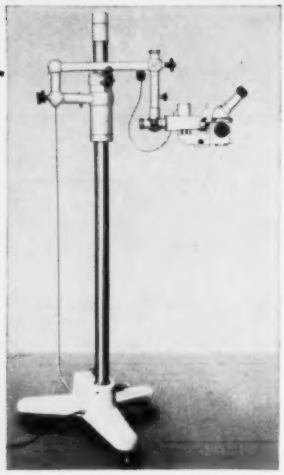
N-1522* Handpiece, CHAYES: No. 2, with single pulley. Can be autoclaved. Stainless Steel $49.25

Special Folder,
"Instruments for Endaural Surgery"
Sent on request.

N-1520
N-1522

Storz Instrument Company

4570 Audubon Avenue — St. Louis 10, Mo.



SHAMBAUGH-DERLACKI

ZEISS OTOSCOPE MICROSCOPE

Has free working distance of 8 inches. Intense illumination parallel with line of vision gives bright field of view for stereoscopic observation, even in deep cavities. Body has quick changer to magnifications of 6X, 10X, 16X, 25X and 40X.

AU-2910—Includes microscope body with magnification changer, clamp-on lever, and dovetails for photographic attachments; microscope objective (f=200 mm.); inclined binocular tube; pair focusing oculars, 20X; movable floorstand, 1650 mm., with counterweight, transformer and power pack for electronic flash; two coupling pieces; 3 spare lamps; plastic cover for microscope. For operation on 110-115 volts, 60 cycles, AC ..... $1912.50.

Set of sterilizable stainless metal shields, for eye pieces and objectives $22.50
Sterilizable muslin sleeve for entire microscope, each 10.00
Complete equipment, as above, including shields and sleeve 1945.00

AU-2910E—Focusing Eyepieces, 12.5X. Extra, per pair 78.00

Photographic equipment for above is available to order.

Ask for your copy of Armamentarium, Vol. II, No. X.

Armamentarium

V. MUELLER & CO.
330 South Honore Street
Chicago 12, Illinois

DALLAS • HOUSTON • LOS ANGELES • ROCHESTER, MINN.

In acute and chronic

Sinusitis

By using the NICHOLS NASAL SYPHON, these cavities may be safely and successfully drained of mucopurulent material, thus affording prompt symptomatic relief. Used and prescribed for more than 40 years. Prove its effectiveness on your most troublesome cases. Just mail the coupon.

Nichols NASAL SYPHON

NICHOLS NASAL SYPHON, INC. MONTCLAIR, N. J.

Please send details of SPECIAL OFFER "S" without obligation

NAME..... M.D.
ADDRESS.....
CITY..... ZONE..... STATE.....

compare this

Beltone

PORTABLE MODEL 10-A

with any other audiometer

Diagnostic hearing tests are made simpler, faster than ever before, thanks to the Beltone Model 10-A Portable Audiometer's outstanding features. Here's why:

1. **Accuracy**—Continuously variable attenuation permits interpolation in single or even ½ decibel steps. Exclusive one tube circuit and single induction coil mean trouble free circuitry.
2. **Complete Facilities for Testing**—Portability lets you take audiometer into the operating room for exacting audio-surgical work. Can be used with the new Beltone Acoustic Bone Probe for determining the functional integrity of the ossicular chain. Audiometer has both air and bone conduction receivers, complex masking calibrated in decibels of effective masking. Receivers are individually equalized.
3. **Ease of Operation**—large, easy-to-read dials allow operator to prepare audiograms up to 33⅓% faster.
4. **Weight**—only 11 pounds, 40% less than an instrument using 3 or more tubes.

Leading physicians, hospitals, and clinics have adopted the Beltone portable audiometer as the *one* audiometer that satisfies their exacting requirements. And its price—only $350—means that the Beltone Model 10-A costs very little to buy, service and maintain. Other Beltone Portable Audiometers, in addition to those shown here, from $295.

Write for FREE Descriptive Booklet

Beltone®

AUDIOMETERS

produced by the Audiometer Division

BELTONE HEARING AID CO.

WORLD'S LARGEST EXCLUSIVE MANUFACTURERS OF AUDIOMETERS AND TRANSISTOR HEARING AIDS

2900 West 36th Street, Chicago 32, Illinois

Other Beltone Audiometers include:

12-A AUDIOMETER

Model 12-A, $395. Identical to Model 10-A, except that it is equipped with a microphone circuit and a meter jack for live voice and recorded speech testing.

15-A AUDIOMETER

Model 15-A Clinical Audiometer, $785. Beltone's finest audiometric instrument. Enables you to do all the speech and pure tone tests necessary for a complete hearing evaluation. Equipped with two channels which may be used independently or mixed and white noise or complex noise masking. Provides inputs for accessories such as tape recorder, microphone, phonograph, warble tone, P.G.S.R. unit, plus all standard audiometric equipment.

Mail coupon today for FREE, illustrated booklet that shows how the Beltone Portable Audiometer can simplify your testing and clinical needs. Specifications and features are thoroughly described. No obligation.

Audiometer Division, Beltone Hearing Aid Co., Dept. 9-135
2900 W. 36th St., Chicago 32, Ill.

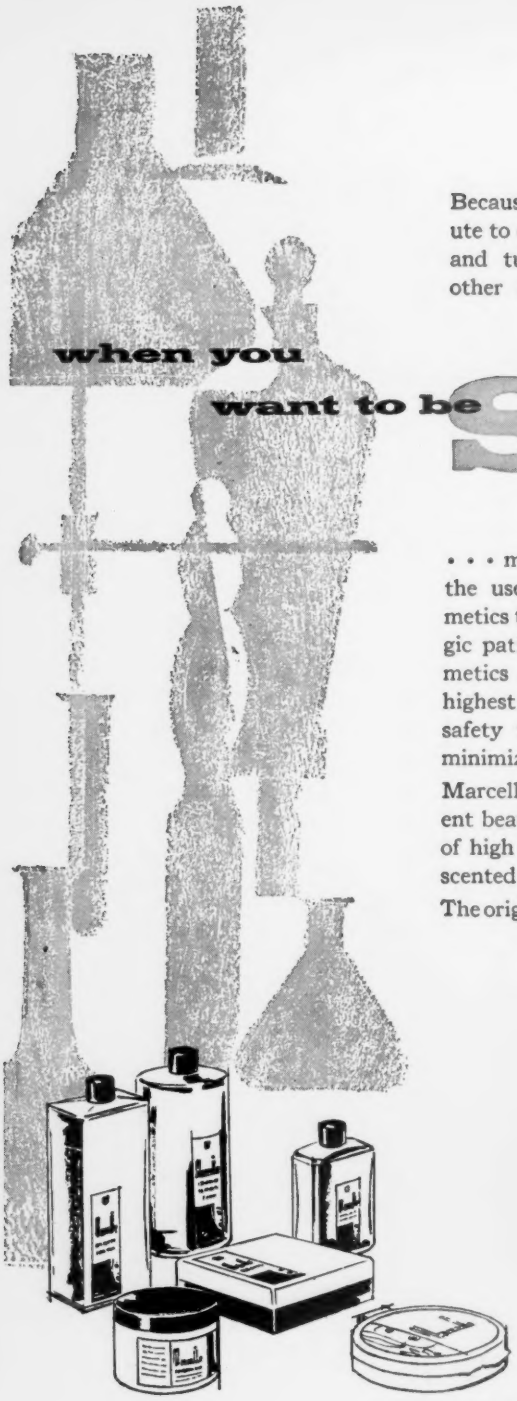
Please rush me, without obligation, your FREE Booklet that shows how I may simplify hearing tests and save money with the Beltone Portable Audiometer.

Name_____

Address_____

City_____ Zone____ State_____

Position_____



Because cosmetics may provoke or contribute to edema of the nasal mucous membrane and turbinates, inhalation difficulties and other respiratory allergic reactions • • •

when you want to be SURE

• • • many physicians routinely recommend the use of Marcelle Hypo-Allergenic Cosmetics to *protect* their treatment of the allergic patient, Marcelle Hypo-Allergenic Cosmetics are compounded according to the highest standards of purity, quality and safety with known allergens and irritants minimized or eliminated.

Marcelle's entire line of more than 40 different beauty preparations in a complete range of high fashion shades is available in either scented or unscented form.

The original Hypo-Allergenic Cosmetics.

Marcelle
HYPO-ALLERGENIC
COSMETICS

FOR SENSITIVE AND ALLERGIC SKINS

For formulae of Marcelle products, testing materials or consultation concerning special cases of cosmetic sensitivity, write to:

MARCELLE COSMETICS, INC., 1741 NORTH WESTERN AVENUE, CHICAGO 47, ILLINOIS

Distributed in Canada by PROFESSIONAL SALES CORPORATION, 2765 Bates Road • Montreal, Quebec, Canada